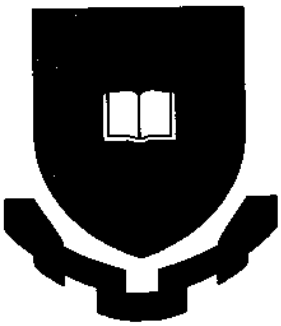
ORAL HEALTH OF CHILDREN WITH GASTROESOPHAGEAL REFLUX

Vivienne M. Linnett BDSc



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I, Vivienne Linnett, of 43 Nadine Street, Graceville, do solemnly and sincerely declare that these Research Project Reports have been composed by myself and have not been accepted in part or in full for another degree. I make this declaration conscientiously believing the same to be true before a Justice of the Peace.

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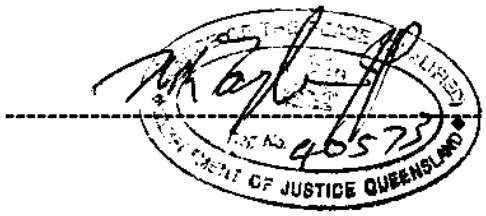

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PREFACE

A few epidemiological studies have shown that dental erosion may occur frequently in children. Changes seen in dental erosion vary from loss of surface characteristics to extensive loss of tooth tissue with pulp exposure and abscess formation. The cause of such tooth loss may be multifactorial, and include combinations of erosion, abrasion and attrition. A multitude of factors may modify the erosion process, including salivary parameters, which may modify oral pH. When dental erosion is diagnosed, it is important to investigate the cause, which is usually due to extrinsic or intrinsic acids. The aim of treatment is to eliminate the cause of acid exposure, and to minimize the effects of acid exposure where it is not possible to remove the acid source, as well as to restore the dentition. The first paper in this thesis, submitted for publication in Pediatric Dentistry, examines the prevalence, clinical manifestations, and etiologic factors of erosion in children. The role of gastro-oesophageal reflux (GOR) is considered including the diagnosis and management, and the relationship between GOR and erosion. The second paper, also submitted for publication in Pediatric Dentistry, examines the oral health of a group of children with GOR comparing them to healthy siblings.

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Oral Health of Children with Gastroesophageal Reflux

DENTAL EROSION IN CHILDREN: A LITERATURE REVIEW

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ABSTRACT

Epidemiological studies have shown that the prevalence of dental erosion in children varies widely between 2 and 57 %. Changes seen in dental erosion range from removal of surface characteristics to extensive loss of tooth tissue with pulp exposure and abscess formation. Symptoms of dental erosion range from sensitivity to severe pain associated with pulp exposure. The etiology of dental erosion is dependent on the presence of extrinsic or intrinsic acid in the oral environment. Extrinsic sources of acids in children include frequent consumption of acidic foods and drinks, and acidic medications. Regurgitation of gastric contents into the mouth, as occurs in gastroesophageal reflux, is the most common source of intrinsic acid in children. A multitude of factors may modify the erosion process, such as saliva, oral hygiene practices, and presence or absence of fluoride. When dental erosion is diagnosed, it is important to investigate and identify the acid source, and to determine if the process is ongoing. The aim of treatment is to eliminate the cause of acid exposure, and to minimize the effects of acid exposure where it is not possible to remove the acid source. Restoration of the dentition involves stainless steel crowns to restore lost vertical dimension, and composite resin for aesthetics.

INTRODUCTION

Erosion is a chemical dissolution of the dental hard tissues by intrinsic or extrinsic acids.[1] In recent years, dental erosion has been increasingly recognized as an important cause of tooth structure loss, not only in adults, but also in children and adolescents.[2-16] Dental erosion may cause tooth sensitivity, altered occlusion, compromised aesthetics, and in severe cases may result in pulp exposure and abscesses. However, the pathogenic mechanisms, diagnostic criteria, and preventive strategies of the condition are still not well established.

The aims of this paper are to review the prevalence, clinical manifestations, and etiology of dental erosion in children, and to provide guidelines on the preventive and restorative options for this condition.

PREVALENCE OF EROSION

There are few prevalence studies of dental erosion and of these, case reports or small sample studies comprise most of the dental literature.[3] (Table 1) In addition, different indices in the diagnosis of erosion are used making comparison between studies difficult.[4, 17]

In the primary dentition, the UK Child Dental Health Survey showed that the prevalence of erosion on palatal surfaces of the primary teeth was 8% in 2 year olds and 52% in 5-year-olds, and the proportion of children exhibiting erosion extending into dentin was 24% in 5 year olds.[5] A study by Millward et al,[8] in children 4-16 years of age, found dentin exposure in 30% of primary molars.

In the permanent dentition the prevalence of erosion on palatal surfaces was 8% in 7 year olds and rose to 31% in 14-year-old children.[5] Children exhibiting erosion extending into dentin was 2% in 15 year olds.[5] In other studies, Bartlett et al,[6] found dentin erosion in 2% of 11 to 14 year olds, and Milosevic et al[7] in a random sample of 14 year old children found 30% had exposed dentin incisally and 8% had exposed dentin on occlusal and or lingual surfaces.

In the primary dentition, it is thought that the reduced thickness of enamel and greater acid solubility contributes to the higher susceptibility to erosion.[4, 16]

Children with cerebral palsy are thought to have an increased prevalence of tooth wear, which has been attributed to both oral parafunctional activity, and softening of the enamel from gastroesophageal reflux.[9]

CLINICAL MANIFESTATIONS OF EROSION

Appearance and distribution of erosion lesions

It is now thought that both lingual and buccal surfaces may be affected in erosion lesions resulting from both intrinsic and extrinsic sources of acid, although intrinsic causes of erosion tend to affect more lingual surfaces.[14] In addition, anatomical factors related to movements of the tongue, lips and cheek may affect the distribution of the erosion lesions. [20] Salivary factors such as pellicle formation appear to affect development of erosion. [21, 22]

Table 1. Prevalence of dental erosion in children

Authors/ year	Number of subjects	Age of subjects (Mean)	Diagnostic criteria	Prevalence of erosion
Millward et al, 1994[8]	101	4 – 16.5 (9.8)	TWI*[18] Dentin exposure	30% of primary molars
Milosevic et al, 1993[7]	1035	14	Dentin exposure	30% incisal 8% occlusal or lingual
Bartlett et al, 1998[6]	210	11 - 14 (12)	Dentin exposure	2% on incisal and palatal of permanent incisors
O'Brien UK survey 1993[5]	2000	7 year olds 14 year olds	Palatal surface erosion	8% 31%
		5 year olds 15 year olds	Dentin erosion	24% primary teeth 2% permanent teeth
Hinds et al[19]	1995	1.5 to 4.5 yrs	Dentin exposure	8% primary teeth

* TWI = Tooth Wear Index

Physiologic tooth wear is normally contributed by a combination of abrasion, attrition and erosion. However in any one individual, each may be seen in differing proportions, complicating diagnosis. For example, enamel softened by erosion is more susceptible to abrasion and attrition.

Erosion usually manifests as concave loss of tooth surface. In contrast, tooth attrition or physiological wearing away of dental hard tissue as a result of tooth to tooth contact, causes incisal or occlusal surface loss of tooth substance, resulting in the formation of facets.[10, 11] In addition, erosion lesions may be distinguished from attrition where defects on opposing teeth cannot be brought into occlusal contact, demonstrating the characteristic "cupping" of erosion.[11]

Erosion may also be distinguished from abrasion, which is the pathologic wearing away of tooth substance by an abnormal mechanical process independent of chewing. [10] Abrasion is more likely to affect buccal or cervical surfaces of teeth, and is often caused by toothbrushing. [13]

The hypothesis of abfraction describes a wedge-shaped defect with a sharp outline at the cemento-enamel junction (CEJ) and is thought to be due to occlusal forces causing microfractures of enamel and dentin at the CEJ.[23] Further research is necessary to determine the role of abfraction in tooth wear. [10]

Initially, erosion may be manifested by a slight loss of surface luster only detectable when the enamel is cleaned and dry. [11] Sensitivity or fracturing of thinned incisal edges may be the first signs of erosion because of its insidious nature.[3, 11] The erosion progresses until the more yellow underlying dentin becomes visible through the thinned overlying enamel.[11, 24] These lesions have a dished out, hard, smooth appearance.

Several different classifications have been used in the literature to describe dental erosion. A set of diagnostic criteria to define dental erosion caused by dietary acids in adults, was proposed by Eccles and Jenkins [14]. In this classification, mild forms of erosion are seen as loss of surface enamel characteristics resulting in smooth glazed surfaces. On labial and palatal surfaces, this may progress to complete loss of enamel with increased translucency interproximally. Loss of enamel on incisal edges gives a grooved appearance. In cervical areas, concavities may be seen in which the width of the lesion exceeds the depth, unlike abrasion lesions. Posterior teeth cusps may show the characteristic cupping or depressions. Amalgam restorations may have the appearance of being raised above surrounding tooth structure. It was noted that attrition or abrasion complicates the diagnosis of the erosive lesions. [14]

The Smith and Knight Tooth Wear Index [21] is used to score degrees of wear in adults from all types of wear: attrition, abrasion and erosion. Each tooth is scored by examining four surfaces: cervical, remainder of buccal or labial surface, lingual or palatal surface and the occlusal or incisal surface.

A classification of dental erosion in children with gastroesophageal disease has been proposed by Aine et al, [15] ranging from grade 0 where there is no erosion, to grade 3 where there is exposure of dentin at the bottom of holes in the occlusal surface. This classification is specific for children with GOR and is suitable for scoring primary, mixed and permanent dentitions. It was found that most children with pathologic GOR exhibited dental erosions of the same type but with varying severity.

Severe dental erosion lesions in children may be seen in figures 1, 2 and 3. These are the result of GOR, and excessive frequent consumption of acidic drinks.

Fig 1A Maxillary teeth of boy aged 8 years who had a history of gastroesophageal reflux due to incompetent lower esophageal sphincter. Erosion grade 3 as described by Aine et al [15] is clearly seen on the primary teeth. Note also the more severe erosion on the palatal/lingual surfaces as distinct from the erosion seen on labial surfaces in Figure 2, where erosion was attributed to consumption of acidic drinks.

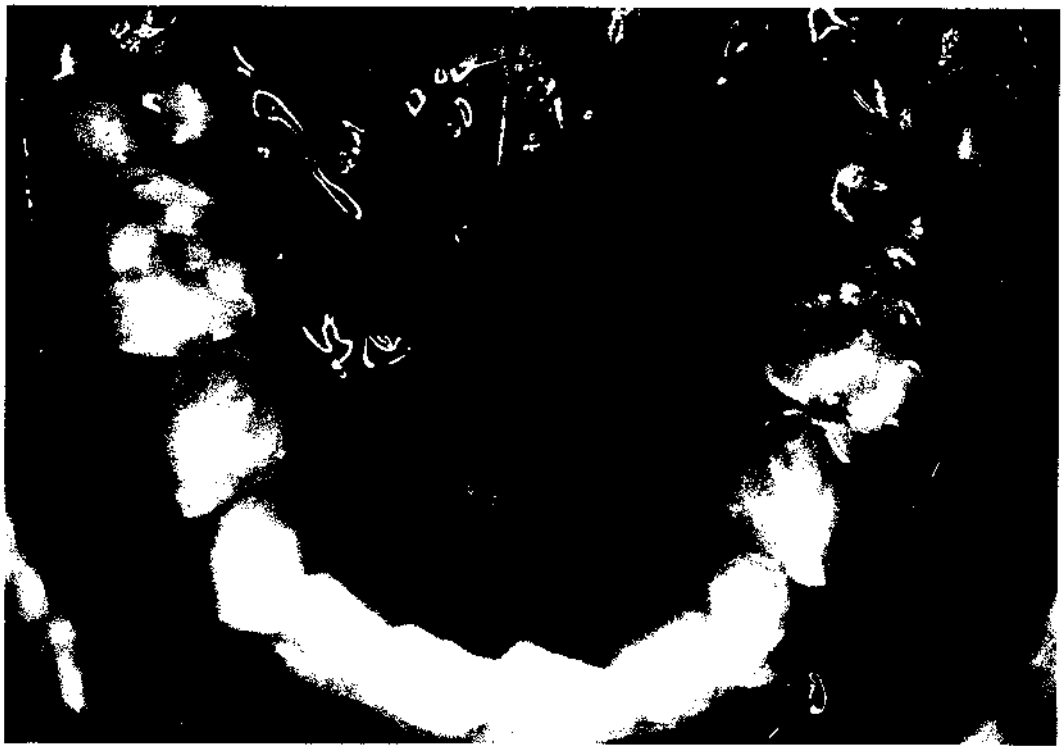

Figure 1B: Mandibular teeth of boy shown in Figure 1A showing grade 3 erosion on the primary teeth.

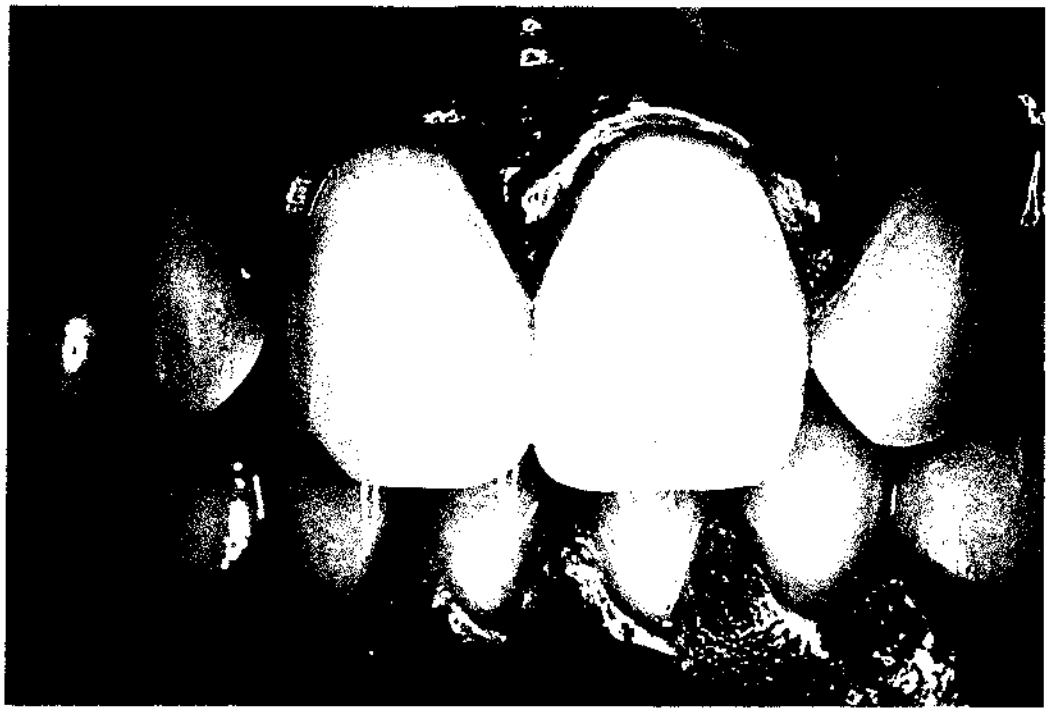

Fig 2: Anterior teeth of a 12 year-old boy who had frequent consumption of acidic drinks. Note the etched appearance of the left central incisor (grade 1 erosion) and the thinning of the incisal edge of the right central incisor (grade 2 erosion).

Fig 3: Mandibular teeth of a 9 year old boy who had frequent consumption of acidic drinks. Note the appearance of the amalgam restorations sitting above the remaining tooth structure, also dentin exposure (grade 3 erosion)[15]

Dental complications of erosion

Dental erosion may cause a number of clinical problems including aesthetics. Severe erosion usually results in enamel fracture, which progresses to shortening of the teeth and loss of occlusal vertical dimension. [16, 25]

Dentin sensitivity and difficulty in eating, is a common problem of dental erosion, particularly if erosion is rapid, and progressive. Rapid loss of tooth structure from dental erosion in children with immature teeth and large pulps are likely to lead to pulpal inflammation and exposures. [26]

ETIOLOGY OF DENTAL EROSION

The underlying etiology of dental erosion is a source of acid, which may be intrinsic or extrinsic, acting on a susceptible tooth. In addition, there are many modifying factors affecting the host which significantly affect tooth susceptibility to dental erosion. Parafunctional habits may also contribute to tooth wear in teeth that have been softened by acid demineralization. [27]

Extrinsic sources of acids

Extrinsic causes of dental erosion may arise from several sources, including those which may be occupational, in medications, or through lifestyle practices. (Table 2) Although not affecting children, occupations involving exposure to acids in the workplace may contribute to dental erosion. Workers in factories in which there are acidic fumes or aerosols involving sulfuric acid, such as in battery factories, and hydrochloric acid, such as in galvanizing factories, have been shown to have a higher prevalence of erosion.[28] Several other occupations have been implicated in increased tooth surface loss, including professional wine tasters, [29] printers[30] and workers in munitions factories. [28] Improper

Table 2. Extrinsic acid sources which have been implicated in erosion

Workplace/ Occupational	Medication	Diet
Acidic environments such as in fertilizer, battery, munitions, printing or galvanizing plants.	Acidic medications eg chewable vitamin C tablets, aspirin.	Frequent consumption of acidic foods eg citrus fruits, pineapple, salad dressing containing vinegar
Swimming in improperly chlorinated pools		Frequent consumption of acidic drinks eg cola, sports drinks, fruit juice
Professional wine tasters		Sucking on citrus fruits
Sporting activities causing dehydration followed by acidic sports drinks		Alcohol eg wine, spirits mixed with acidic soft drinks

monitoring of pH in gas chlorinated swimming pools has been reported to be the cause of dental erosion in competitive swimmers. [31, 32]

Dehydration following sporting activities may contribute to erosion when reduced salivary flow causes decreased buffering. This may be exacerbated by consumption of acidic drinks such as sports drinks, fruit and soft drinks.[26]

Chewable Vitamin C preparations may cause erosion when consumed frequently and left in direct contact with the teeth. [33, 34] Aspirin when chewed daily over extended periods has been reported to cause erosion in children. [35] Erosion has been associated with the consumption of citrus fruits, low pH carbonated drinks, cider vinegar and sports drinks.[36] Consumption of alcohol, for example wine and when acidic carbonated drinks are used as mixers, may also contribute to erosion. [37]

Titratable acidity of foods and drinks

The pH of the oral cavity affects the solubility of dental tissues. The solubility increases by a factor of seven to eight with each decrease of pH by 1 unit when oral pH decreases from normal (pH 6.5) to acidic. [38] As the critical pH at which enamel dissolution occurs is 5.5, acidic products with a pH below 4 will result in erosion. [39]

In relation to enamel dissolution, the actual H^+ of acidic dietary substances available to interact with the tooth surface (or titratable acidity) is more important than actual pH. [40] Modifying effects of other constituents of food and beverages such as calcium, phosphate and fluoride concentration may also be exerted. Factors including the acid type and physical and chemical properties may affect the salivary clearance rate from the mouth.[40] These effects have been shown in the studies of Lussi et al [41] who found that erosive capacities of the different test substances were significantly associated with their titratable acidity, pH, phosphate content and fluoride content. Furthermore, a high

consumption pattern of acidic beverages such as cola drinks may present a higher risk of causing erosion than based solely on their chemical properties.[40]

Types of acidic foods and drinks associated with erosion

Consumption of acidic foods and beverages has been shown to contribute to dental erosion. In a clinical trial which investigated the effects of acidic beverages on human teeth in a group of dental students, the effect of daily ingestion of different amounts of acidic beverages on macroscopic and microscopic changes in the labial surface of maxillary anterior teeth was examined.[42] The students were divided into groups, drinking either orange juice, grapefruit juice or carbonated cola, and subdivided into groups of five who drank either 6,12, 18, or 24 ounces of the juice or carbonated beverage per day. A group of 10 students served as controls and refrained from ingesting all forms of citrus fruits and carbonated beverages. The study reported that the first appearance of any microscopic alteration of the enamel surface occurred between the fourth and sixth weeks, and that all experimental groups were found to have some alteration of surface enamel. Orange juice was found to cause less erosion than grapefruit juice or carbonated cola beverage. However, even within the high consumption groups, some students did not experience any detectable erosion, suggesting that there may be biological modifying factors present.[42]

Sports drinks have also been evaluated in other studies. Citric acid is frequently included in sports drinks for its refreshing taste, but has been found to be highly erosive. The demineralizing effect of citric acid is exceptionally great because its chelating effect on enamel calcium continues even after the pH rises.[36]

Frequency of consumption

Jarvinen et al[36] found a strong association of dental erosion, and the consumption of citrus fruits more than twice a day, soft drinks daily, and apple vinegar or sports drinks once a week or more.

Other studies have also shown an increase in the mean frequency of consumption of fruit drinks, carbonated beverages and fruit juices were each associated with an increase in the severity of erosion. Of note was the finding that bedtime consumption of fruit juices was strongly associated with the most severe cases of erosion, [8] suggesting that the erosive potential of fruit juices was probably the highest when salivary flow is the lowest.

Intrinsic Sources of Acid

Sources of intrinsic acids causing dental erosion include the propulsion of gastric contents into the mouth. This occurs in disorders such as anorexia nervosa and bulimia[43, 44] and gastroesophageal reflux.[45]

Dental erosion has been observed in disorders associated with chronic vomiting, with persistent regurgitation or gastroesophageal reflux, or with protracted rumination. Conditions in which these occur include disorders of the upper gastrointestinal tract, specific metabolic and endocrine disorders, medication side effects, and drug abuse, as well as psychosomatic disorders (stress induced psychogenic vomiting, anorexia nervosa and bulimia nervosa, rumination).[46]

Gastric dysfunction is one of the principal risk factors associated with dental erosion. Patients reporting symptoms such as vomiting once or more per week, experiencing acid tastes, belching, heartburn, stomach-ache, or pain on awakening, have 31 times higher incidence of dental erosion when compared to controls.[36]

Clinical manifestation of erosion does not usually occur until gastric acid has acted on the dental hard tissues regularly several times a week for a period of at least 1-2 years.[43]

Gastroesophageal reflux

Definition

Gastroesophageal reflux (GOR) is defined as the involuntary passage of gastric contents into the oesophagus.[47] It may be primary, due to anatomical or physiological abnormalities, or secondary, due to conditions such as anxiety, intolerance to certain foods (heavily spiced or fatty meals) and drinks (such as alcohol),[17] metabolic disorders, and reactions to certain drugs. Other medical problems such as infection, intestinal obstruction, intestinal atresia, or pyloric stenosis and intracranial pathologies such as hydrocephalus, neoplasia, or a subdural haematoma have also been implicated in GOR.[47]

Pathophysiology of GOR involves several factors including transient lower oesophageal sphincter relaxation, increases in intra-abdominal pressure, and decreased lower oesophageal sphincter tone. Reflux frequency is affected by posture/gravity, and increased gastric volume. Duration of reflux is influenced by oesophageal clearance which is dependent on gravity and peristalsis (which determine bulk clearance) and saliva, which is responsible for acid washdown and neutralization.[48]

Medical Diagnosis of GOR

When the clinical history is compatible with symptomatic GOR, a variety of diagnostic studies are available to evaluate the extent of the reflux and its relationship to the patient's symptoms.

In children, pathologic GOR often produces more than one symptom in the same patient. The most common symptom in infants is recurrent vomiting. Failure to thrive caused by caloric deprivation from repeated emesis is the most common manifestation during the first 2 years of life.[49] In contrast to normal children, those with symptomatic GOR have more refluxing episodes when asleep. Children without symptoms rarely reflux in the supine position, in contrast to those with symptomatic GOR, who commonly reflux in both the supine and upright positions.[50]

Repeated emesis with periodic aspiration of gastric contents in infants and young children can produce recurrent bronchitis or pneumonia during sleep, when swallowing and oesophageal clearance of acid are least efficient.[51] Any infant or child who has recurrent pulmonary infections for which a cause cannot be established should undergo evaluation for GOR.[52] Prolonged duration of reflux during sleep has correlated closely with the presence of reflux induced respiratory symptoms.[53]

Several studies suggest that reflux of small amounts of acid into the mid or upper oesophagus may stimulate vagal reflexes and produce reflux laryngospasm, bronchospasm, or both, which may accentuate the symptoms of asthma.[54] A lowering of the threshold for bronchoconstriction may be caused by a reflex triggered by acidification of the oesophageal mucosa.[55] It is difficult to document a diagnosis of reflux-induced asthma; however oesophageal pH monitoring often shows a close association of GOR with acute wheezing or coughing episodes in children with asthma. It is apparent that a large number of children with asthma have significant GOR regardless of the use of bronchodilator therapy. Additionally, most bronchodilator medications used for treatment of children with asthma tend to decrease the lower oesophageal sphincter pressure, which has the effect of increasing likelihood of reflux and aspiration.[56] Infants with GOR

frequently have respiratory symptoms such as apnoea, and bradycardia, pneumonia, cyanosis, or stridor. These patients are often treated extensively for their respiratory symptoms with minimal clinical improvement until diagnostic evaluation of therapy for GOR is carried out.[49]

Repeated acid reflux into the lower oesophagus frequently causes oesophagitis with chronic inflammation of all layers. For infants with reflux oesophagitis, crying is a common problem, whereas for older children heartburn is the most common symptom.[49] Diagnosis of GOR may be made by pH monitoring using an indwelling pH probe in the oesophagus over 18-24 hours and is considered the gold standard for documenting acid GOR.[57] It is generally recognized by gastroenterologists as a pathological level of reflux when the pH is below 4 for more than 4% of the total time (2% at night and 6% during the day).[57] The reflux index is the percentage of time when the lower oesophageal pH is below 4. In general, children with a reflux index of 5-10% are considered to be mildly affected, 10-20% moderately affected and greater than 30% as severely affected. Mild or moderate reflux may be controlled by conservative or pharmacological treatments while the severe cases are more likely to require surgical management.[47]

Endoscopy is also particularly useful in the diagnosis of reflux. The oesophageal mucosa is examined for signs of inflammation such as granularity, bleeding to touch, ulceration, sloughing, exudate and stricture.[58] Histologically the criteria used for reflux change are elongation of papillae to two-thirds of the mucosal thickness, or basal hyperplasia to at least three to four layers thick. Microscopic findings also include ulceration, inflammation, fibrosis, and the appearance of columnar epithelium.[58]

Management – Treatment of Gastro-oesophageal Reflux

Infants with uncomplicated GOR may be diagnosed on the basis of history and examination alone, and often respond to a regimen of fairly simple measures, such as antacids, positioning, and simple dietary advice. If these measures have failed to improve symptoms after 4-6 weeks, and secondary causes of GOR have been excluded, then cisapride (*Prepulsid*, Janssen-Cilag Pty Ltd), a prokinetic drug is used.[59] This is a non-dopamine receptor blocking agent, which acts by enhancing acetylcholine release in the gut, thereby increasing gastrointestinal motility and improving antroduodenal coordination.[60]

If the above regimen fails to show any benefit after a further 4-6 weeks, lower oesophageal pH monitoring studies are indicated. If GOR is confirmed by pH monitoring, treatment is based on the severity of GOR, and the presence of complications.[47]

Management – Mild-Moderate GOR

In patients in whom pH monitoring confirms mild (5-10%) or moderate (10-20%) GOR, treatment with general measures and cisapride is continued for a further period of up to 3 months.[47]

Management – Severe GOR

If pH monitoring indicates severe GOR (over 20%), an H_2 antagonist such as cimetidine (*Tagamet*-Smith Kline and French) or ranitidine (*Zantac*- Glaxo Australia Pty Ltd) is added to prevent oesophagitis while continuing the regimen of cisapride and the general measures as outlined above. If there were clinical indicators of oesophagitis, endoscopy would be carried out before commencing the H_2 antagonist therapy. Omeprazole (*Losec*-

Astra Pharmaceuticals Pty Ltd), a proton pump inhibitor, is also effective in treating GOR related oesophagitis.[47]

A follow-up endoscopy is done 2-3 months after commencement of treatment. If the mucosa has healed, H_2 blockers or omeprazole may be discontinued, but prokinetic agents, antacids, and general antireflux measures should be continued for a prolonged period. If there is no evidence of healing, treatment is continued for a further three months, ensuring that anatomical or other problems have been excluded.

Surgical treatment of GOR (usually a Nissen fundoplication) may be inevitable if full medical treatment has failed, or if there is an oesophageal stricture or Barrett's oesophagus at initial diagnosis.[47]

Prevalence of GOR

Prevalence data on GOR have been difficult to obtain due to the wide variety of clinical presentations.[46] Most individuals experience occasional episodes of reflux, and the point at which normal physiology develops into pathosis is difficult to determine.

The classic symptoms of heartburn and regurgitation are well known to the majority of the population. Nebel,[61] found that 7% of normal individuals can be expected to experience heartburn daily and that a total of 36% of normal subjects have a symptom consistent with heartburn at least once a month.

Recent research indicates that reflux affects 7% of the population on a daily basis and more than 30% every few days.[62] Most of this is asymptomatic but there is a wide spectrum of disease and severity of reflux into the mouth. Sometimes erosion of the dental tissues may be the first sign that reflux is occurring.[4]

The recognition that clinical manifestations of GOR include various symptoms other than heartburn suggests a prevalence considerably higher than previously estimated. In up to

50% of patients with noncardiac chest pain,[63] 78% of patients with chronic hoarseness,[64] and 82% of patients with asthma,[56] an association with GOR may be noted.

The prevalence of GOR in the paediatric population is not known.[65] Children with neurological impairments such as cerebral palsy have significantly higher levels of GOR than 'normal' children.[66]

Clinically important GOR is often found in children with recurrent lower respiratory tract symptoms.[67] In children with recurrent respiratory disorders Buts et al[68] reported a 61% incidence of reflux.

Dental Erosion and GOR

The relationship between GOR and dental erosion is supported by findings in anorexia and bulimia nervosa in which similar effects are seen on the teeth. Wear patterns on the palatal surfaces of the upper incisor teeth in anorexia and bulimia nervosa are similar to those seen in patients with GOR.[69]

Since the acidity of the stomach may be below pH 1, frequent regurgitation or vomiting may cause erosion. Refluxed acid attacks the palatal surfaces of the upper incisors initially, and if the condition continues other teeth may be involved

The prevalence of dental erosion in patients with reflux has been reported by several authors. Meurman et al[70] examined 117 patients with reflux disease and reported that 28 patients (24%) had dental erosion. In these studies it was also found that the number of patients with low salivary buffering capacity was higher among those with erosion than those without. Bartlett et al,[71] examined 36 patients who were investigated because of their palatal dental erosion and found that 23 (64%) had gastroesophageal reflux. They concluded that patients presenting with palatal dental erosion should be investigated for GOR, even in the absence of clinical symptoms of reflux.

In children, the literature is limited on the role of GOR in erosion. Taylor et al[2] reported an 8-year-old female with extensive loss of enamel on all surfaces of her remaining primary teeth, who on investigation, was found to have asymptomatic gastroesophageal reflux.

Aine et al,[15] found erosive lesions in 15 of 17 children aged 22 months to 16 years with pathological GOR. Seven children out of 15 had dentin exposure. It was concluded that loss of dental hard tissue is an important sign of pathological GOR and that dentists become capable in screening and identifying clinically important silent GOR.[15]

Gudmundsson et al[79] investigated 14 adults and children with erosion using 24-hour pH monitoring in the esophagus and oral cavity. Although no changes were found in oral pH in a total of 339 reflux episodes, significantly more patients with erosion had low salivary buffer capacity compared to controls.

O'Sullivan et al[72] examined children attending hospital clinics with symptoms of GOR whose reflux index was 10% (ie. moderate to severe) or more. Evidence of erosion was seen in 17% of children with only one child having erosion that involved dentin. In all children with erosion, only the primary dentition was affected, generally the palatal surfaces of the maxillary primary incisors.

Pattern of erosion

Gastric or dietary acid entering the mouth results first in exposure of the upper anterior teeth. The tongue, which binds the acid, touches the upper teeth. Erosion is severest on those palatal surfaces touched by the tongue.[18] The rinsing effect of saliva on the upper teeth is weak. The buffering of saliva on plaque pH is weaker on the surfaces of the upper than of the lower incisors.[73] If flow rate of saliva is low, its buffering capacity is even worse and the risk of erosion is particularly high.[36] In patients with reduced salivary

flow, salivary clearance time is also increased and acidity in the mouth remains high for longer than in those with normal salivary flow. The rarity of severe erosion on buccal surfaces may be a result of rapid passage of acid on buccal surfaces and of rinsing of these surfaces by saliva from the small glands in lips and buccal mucosa.[20] Several articles have suggested a relationship between palatal dental erosion and gastro-oesophageal disturbances.[2, 45]

It is thought that erosion occurs when the pH is less than 4 for at least 5% of each 24 hour period.[65] Because the pH of the gastric contents is consistently less than 1, reflux into the oral cavity overwhelms local buffering, resulting in surface enamel dissolution. Severity of symptoms ranges from opacities or white spots to a flattening of the cusps and eventual dentine exposure.[2, 15] If this is a slowly progressing condition, narrowing of the pulps may be seen due to the deposition of secondary dentine. If the progression is more rapid, the pulps may be clearly visible through the occlusal surface due to the loss of hard tissue, and eventually exposed, resulting in pulpal infection and abscess formation.[2] Both primary and permanent teeth may be affected.

The typical distribution of dental erosions in anorexia and bulimia involve the lingual surfaces of the maxillary teeth and facial surfaces of the maxillary incisors and canines.[74] Regurgitation erosion occurs on the exposed surfaces of the teeth rather than the inaccessible plaque retentive areas where caries develops.[75]

Factors modifying the erosion process

Individual responses may also influence erosion. These include the manner in which the erosive fluid is taken into the mouth, the tooth surfaces that come into contact with the fluid, and the duration of contact with the teeth. This in turn is influenced by swallowing habits, motions of the lips and cheeks, and access to saliva. Other host factors are also

considered to modify the erosion process, such as the buffering capacity of the saliva, the chemical and physical properties of enamel and the shape and contour of the teeth.[42]

Saliva

Of the factors modifying the erosion process, saliva is probably the most important as it is known to have protective properties against dental erosion,[40] but the nature of this role is not fully established.[76]

A number of previous studies have examined various salivary parameters and their relationship to dental erosion. Saliva forms the pellicle that protects enamel from acid demineralization. [21, 22, 76] The thickness of pellicle varies within different areas of the mouth and this may influence the sites and severity of erosion. [77] Unstimulated salivary flow rate[36, 78] and the buffering capacity[79] have been directly associated with dental erosion. A direct relationship has been demonstrated between reduced salivary flow rates and oral clearance of dietary acids.[80] In addition, salivary bicarbonate levels are correlated with salivary flow rate; therefore, when flow rate is low, salivary pH and buffering capacity is lower.[80]

Significantly lower unstimulated saliva secretion rates have been found in individuals with erosion compared to controls, although the stimulated secretion rate was normal.[78] In the study by Jarvinen et al,[45] three patients out of seven with erosion had a distinctly reduced salivary secretion rate. Low unstimulated salivary flow rate was found to be a significant factor for erosion risk.[36] Decreased salivary flow due to dehydration may also contribute to reduced protection by saliva from intrinsic and extrinsic acids.[27]

Gudmundsonn et al,[79] found salivary buffer capacity significantly lower in patients with erosion than controls. O'Sullivan and Curzon[81] also found that individuals with erosion had a low buffering capacity.

Oral Hygiene Practices

Dental erosion is frequently seen in individuals with a high level of oral hygiene.[4] The reason for this may be that abrasive-containing toothpastes can remove pellicle and its protective effect from acids. Initially, the demineralization occurring after acid consumption is reversible, and may be remineralized by salivary minerals. [82] However, enamel and dentin initially demineralized by acid may be easily removed by toothbrushing in a process of abrasion which accelerates the erosion. Hence, the practice of toothbrushing immediately after consuming acidic beverages may increase tooth loss.[83] The alternative of brushing before meals is also likely to have similar effects in that the removal of salivary pellicle[82] will render the enamel surface more susceptible to acid attack during the meal.[84]

Fluoride

It has been shown that the addition of fluoride to acid solutions decreases the amount of erosion in animals.[85] Furthermore, softening of enamel by cola beverage in vitro may be inhibited by high concentration fluoride varnish, [86] and less tooth wear occurred when a fluoride dentrifice is used than when a non-fluoride dentrifice is used in vitro.[87] Fluoride exposure from water fluoridation and in supplement form in the first 12 years of life appears to confer some resistance to excessive tooth wear from acid erosion in adulthood, as it does for resistance to demineralization by dental caries.[88] Application of 2000 ppm sodium fluoride solutions immediately before toothbrushing significantly reduces abrasion of eroded dentin in vitro.[89] Application of titanium tetrafluoride may be effective in prevention of erosion in patients with frequent vomiting or gastroesophageal reflux.[90]

EFFECTS OF INCREASED ACIDITY ON PLAQUE ECOLOGY

Changes in S. mutans Levels

The major group of cariogenic bacteria is now identified as mutans streptococci. The species *S. mutans* and *S. sobrinus* are the most commonly isolated in human dental caries.[91] *S. mutans* is highly aciduric and it may be that the acidic environment found in reflux would favor its colonization. When gastric contents are brought to the upper gastrointestinal system, either self-induced vomiting or by GOR, the resulting changes in the oral environment could be similar in both groups. A few studies have investigated possible changes in the levels of mutans streptococci subsequent to gastric acid presence in the mouth.

Bretz et al,[92] reported that the prevalence and levels of mutans streptococci tended to be higher in bulimics than in non-bulimics. Also, the bulimics had significantly higher levels and higher prevalences of *Streptococcus sobrinus* when compared to non-bulimics. It was suggested that vomiting in bulimia might favor aciduric bacteria and make the colonization of *Streptococcus sobrinus* easier.[92] In a study by Holtta et al,[93] it was found that despite the likely acidic oral environment in reflux children, selection towards a higher occurrence of salivary *Streptococcus sobrinus* was not found.

Isolation frequency of mutans streptococci in a gastroesophageal reflux group of children with mean age of 9.3 was 75%.[93] In a healthy Finnish child population, the prevalence of mutans streptococci was 46% in 5 year olds and 83% in teenagers.[94, 95] In another study of Finnish children with a high caries experience and mean age of 8.3 years, 62% harbored mutans streptococci.[96] This would tend to indicate that the reflux group had a higher prevalence of mutans streptococci. However, unfavorable diet is known to favor

the colonization of mutans streptococci and the role of diet was not considered in this study.

In a study of patients with gastric symptoms by Jarvinen,[45] it was found that there were no particular differences in the salivary microbial counts between patients with either increased acid output, normal acidity or alkaline reflux.

Depending on the strength and frequency of erosive challenges, no plaque microorganisms can tolerate the low pH seen in erosion.[97] Acidic foods and drinks may have pH values below 3, and the pH of gastric contents may be below 1. Cariogenic mutans streptococci cease to metabolize at pH values below 4.2.[98] This explains why erosion and caries are usually not seen on the same tooth surfaces.[97]

Helicobacter Pylori

Helicobacter pylori (H. pylori) is a gram-negative, microaerophilic, motile bacterium, especially adapted to life in the human stomach. It was initially classified as *Campylobacter pyloridis.*[99] The presence of *H. pylori* is strongly associated with histologically proven chronic gastritis and peptic ulcer and is recognized as a risk for gastric cancer.[100] The microorganism may be transmitted orally, and has been detected in dental plaque, saliva and faeces.[101]

H. pylori may be present in the oral cavity as a consequence of gastric reflux. It is probably transient rather than a routine member of the microflora.[102] In most cases, patients with positive oral specimens have positive gastric biopsies, but many patients with gastric *H. pylori* do not exhibit oral co-infection.[102] Most authors consider dental plaque as the second natural reservoir of *H.. pylori*, which could explain the frequent relapse seen after ulcer therapy, but its presence is not associated with any specific oral disease.[102]

DENTAL MANAGEMENT OF TOOTH EROSION

Immediate Management

Patients who have erosion of the palatal surfaces of maxillary teeth should be investigated for a history of reflux symptoms. If periodic symptoms of heartburn, epigastric pain or regurgitation are experienced, with or without recurrent hoarseness and laryngitis, early referral to a gastroenterologist is desirable.[71] A careful history with a thorough medical history is important to identify any causative medical conditions.[13] This should be followed up with a diet history of 7 days to elicit possible dietary causative factors. Monitoring wear is vitally important, so study models and photographs should by taken.[13, 103]

If the acid is dietary in origin, advice must be centered on decreasing the consumption of acidic foods and drinks, and confining the intake of acidic foods and drinks to meal times, and never drinking or eating juice or fruit before bedtime.[13]

Acidic drinks should be swallowed immediately and not 'swished' around the mouth. Finishing a meal with something neutral or alkaline may be beneficial. Eating foods with a high content of calcium, phosphate or lipids or buffering substances may reduce erosive potential. Milk or cheese may help to counteract the erosion resulting from acidic drinks.[104] Patients should be instructed to avoid brushing immediately after consuming acidic food or drink, as this is likely to accelerate abrasion.[83]

A daily neutral sodium fluoride mouthrinse or gel to combat acid damage and control pulpal sensitivity may be prescribed.[87] Acidulated phosphate fluoride should be avoided because of its obvious acidity. Custom fluoride trays may be used but appliances covering teeth must be used with extreme caution if there is a chance of acid being

trapped beneath them.[26] Sugar free chewing gum to stimulate saliva flow may also be advised.[105, 106]

Restorative Management of Tooth Erosion

It is a widely held view that restorative treatment is unwise while erosion is ongoing. The approach of not providing treatment until erosion has ceased is unrealistic.[16]

Many children and adolescents do not comply with dietary advice. Restorative treatment becomes necessary to protect areas of exposed hypersensitive dentine, and where there is risk of tooth fracture or pulpal exposure. Restorations should aim to protect and conserve remaining tooth structure, resolve symptoms of pulpal sensitivity, improve aesthetics and stabilize the occlusion.[16, 26]

The Primary Dentition

Treatment of erosion in the primary dentition is limited by patient compliance, inadequate enamel, and insufficient coronal tissue to provide successful adhesive restorations. Although it is possible to build up worn primary incisors with composite resin, in practice this is seldom done as most primary incisors are close to exfoliation when erosion is diagnosed. Provided worn teeth remain symptom free, they are left unrestored until they are exfoliated. If symptoms occur, these teeth are usually removed.[16]

For posterior primary molars, placement of extra-coronal stainless steel crowns is frequently the only way of providing relief of symptoms and protection from further wear and to maintain the tooth until it is due to exfoliate.[16]

The Permanent Dentition

Enamel loss on anterior teeth may best be restored with composite resin. The material performs best when placed in bulk and offers excellent aesthetics. On the palatal aspect,

however, composite is difficult to apply and easily fractured. It may be necessary to restore the incisal aspect with composite resin and place a more durable restoration on the palatal aspect. Materials suitable for this purpose are yellow gold and nickel-chromium alloys, fabricated as veneers and cemented to the palatal surfaces of worn incisors.[16] Using conservative adhesive techniques, surface active composite luting agents are used in conjunction with sand-blasted nickel-chromium alloy. A similar technique may be used for posterior teeth where nickel-chrome onlays are cemented to restore the occlusal surfaces of eroded molar teeth. In both anterior and posterior teeth, no tooth preparation is required, conserving the maximum amount of tooth structure.[16]

FUTURE STUDIES

More research should be initiated to identify children at risk of erosion so that effective preventive strategies can be initiated. Research into the relative erosive potential of common drinks and foods would enlighten the public as to what foods may damage the teeth. In addition future research into the roles of saliva and medical conditions in the pathogenesis of the erosion lesions may help to further the understanding of this complex condition

CONCLUSIONS

1. Dental erosion is caused by exposure of the teeth to frequent consumption of acidic drinks or foods, or by reflux of gastric acid into the mouth.
2. Clinical manifestations of dental erosion include sensitivity and chipping of incisal edges with consequent poor aesthetics.

3. Children presenting with dental erosion should undergo thorough evaluation to identify source of the acids causing the erosion. In the case of gastroesophageal reflux, medications may be necessary. In addition, the erosive potential of acid may be decreased by dietary alterations, fluoride supplementation and restorative care.

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LIST OF TABLES AND FIGURES

ORAL HEALTH OF CHILDREN WITH GASTROESOPHAGEAL REFLUX.

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ORAL HEALTH OF CHILDREN WITH GASTROESOPHAGEAL REFLUX.

ABSTRACT

The aim of this study was to compare the dental health of children with gastro-oesophageal reflux (GOR) with a healthy control group. Dental examinations were conducted for 52 children (31 boys and 21 girls) with a clear history of GOR. For every subject enrolled in the study, a healthy sibling subject was randomly recruited from the respective families, giving a control group of 52 subjects. Medical histories were obtained from medical records and from parents. Dental and dietary histories were obtained form parents. The teeth were examined for erosion, dental and enamel hypoplasia. *Streptococcus mutans* levels were analyzed using a commercial culture kit. Results show the difference in the prevalence of erosion by teeth was found to be statistically significant between GOR patients (14%) and controls (10%) ($p<0.05$). No differences were found in the number of primary teeth showing erosion between study patients and controls, (20% in both groups). However there was a statistically significant difference in the number of permanent teeth with erosion in that GOR patients had erosion in 4% of permanent teeth compared to 0.8% in the control group ($p<0.05$). When the degree of erosion was compared between the groups, patients in the GOR group had severe erosion in 43% of teeth compared to 9% of controls ($p<0.05$). *Streptococcus mutans* was isolated in 42% of GOR patients and 25% of controls. These figures approach significance ($p = 0.06$). DMF values were higher in GOR patients compared to controls, with study patients showing greater numbers of teeth affected (9.7% compared

to 6.2%, $p < 0.05$). In conclusion it was found that children with GOR had more dental erosion of greater severity, and more caries than healthy control siblings.

INTRODUCTION

Gastroesophageal reflux (GOR) is defined as the involuntary passage of gastric contents into the oesophagus occurring when there is failure of the lower oesophageal sphincter to provide a barrier between the oesophagus and the stomach.[1] Normal physiologic reflux of gastric fluid into the oesophagus with rapid clearance occurs in both children and adults and is of little clinical significance. Symptoms may occur when larger volumes are refluxed frequently.[2] Reflux is considered pathological when it occurs more than normal or when complications arise.[1]

It has been shown 7% of healthy adults may experience reflux daily, and 36% have symptoms monthly. [3] In children, the prevalence of GOR is not known.[4] Vomiting is a frequent presenting symptom in GOR. Other symptoms include heartburn, retrosternal burning discomfort and waterbrash.. There are a number of other clinical signs that fall broadly into two groups. There may be symptoms related to oesophagitis including haematemesis, anaemia, and chest pain. Respiratory complications may occur that are related to aspiration of gastric contents, such as a nocturnal cough, asthma, bronchitis, hoarse voice, pneumonia, apnoea, or stridor. Clinical manifestations of GOR include feeding difficulties and failure to thrive.[1,2]

In children with suspected GOR, a clinical history is essential to establish the nature of symptoms, and associated respiratory or failure to thrive manifestations. Diagnosis by extended oesophageal pH monitoring over 18-24 hours has been considered the gold standard for documenting GOR since its introduction in 1974 by Johnson and DeMeester.[5] However, it is an invasive procedure. A 'reflux episode' occurs when the lower oesophageal pH falls below 4. The reflux index is the percentage time of the study during which the lower oesophageal pH is less than 4. As a general guide, a mild reflux

index (5-10%) or moderate (10-20%) may be controlled with medication, whereas a severe reflux index (over 30%) may require surgery. [1] Endoscopy is useful for examining the oesophagus for macroscopic signs of inflammation followed by histologic confirmation. [1]

As refluxed gastric contents have a pH between 1 and 3, a number of studies have demonstrated that vomiting of acidic gastric contents can lead to dental erosion. [6-17] Dental erosion is a chemical dissolution of the dental hard tissues in a process which does not involve bacteria.[18] Bargen and Austin and later Holst and Lange [19] first reported an association between dental erosion and gastrointestinal disorders. [20]

Although there have been few studies into the association of GOR and dental erosion, particularly in children, individuals exhibiting gastric symptoms have been shown to be at risk of erosion. [21] The prevalence of erosion in children with GOR varies widely with one study reporting erosion in the majority (88%) of children with GOR, [22] and another study reporting erosion in only 17% of cases.[23] In adults, prevalence varies from 16% [24] to 64%.[25]

The aim of this study was to investigate the oral health of children with GOR compared to healthy siblings.

SUBJECTS AND METHODS

Study Subjects

This study was approved by the Human Ethics Committee of the University of Queensland (Appendix 1). Signed informed consent was obtained from each parent (Appendix 2). Children for the study group were obtained from subjects attending the Gastroenterology Department at the Royal Children's Hospital, Brisbane for investigation of reflux. Sixty subjects living in the Brisbane area who attended during early 1999 were

approached. Of these, 52 (31 boys, 21 girls) attended for the dental examination, giving a participation rate of 87%. Subjects attending the Gastroenterology Department from country areas were excluded due to difficulties of distance. All subjects had an endoscopy at the hospital diagnosing their reflux. The ages of children when they attended for the dental examination at the University of Queensland Dental School ranged from 18 months to 15 years. Age of diagnosis of reflux and commencement of medical treatment as obtained from hospital medical charts ranged from 17 months to 12 years.

Control Subjects

Fifty two healthy siblings of study children were also invited to attend for the dental examination and included in the study as the control group (27 boys, 25 girls). The ages of control children when they attended for the dental examination ranged from 17 months to 16 years.

Medical History

A detailed medical history was obtained from the parent during interview prior to the dental examination. Additional medical details were obtained from medical records. The interview obtained details of perinatal history, gestational age, birthweight, postnatal and current medical history. Details of medications both past and present were ascertained, as well as history of the diagnosis and treatment undertaken for the GOR.

Dental History

Details of dental history were obtained from the parent during interview with regard to past dental treatment undertaken, systemic fluoride exposure through residing in a water fluoridated area, use of fluoride supplements, and the presence of any tooth sensitivity to

hot or cold foods and drinks. Oral hygiene habits were ascertained including the number of times per day brushing was carried out, and the type of dentrifice used. These were recorded on the examination form (Appendix 2).

Dietary History

Details of feeding and diet history and frequency of consumption of acidic drinks were obtained during the interview. In addition, parents were asked to complete a 3-day diet diary for their child/children, recording all food and drinks consumed over three consecutive days including one weekend day (Appendix 3). When completed, the diet charts were returned to the Dental School in stamped self-addressed envelopes. This diet diary was examined for the number of times per day each child consumed sugar, both retentive and in solution, and the number of acid exposures per day. Acid exposures included for analysis were in the form of fruit juice, carbonated soft drinks, chewable vitamin C tablets, sports drinks, and citrus fruit consumption.

Dental Examination

The dental examinations were carried out in the Paediatric Clinic at the University of Queensland School of Dentistry, utilizing a dental light, mirror and probe. The results were recorded on standardized data forms. (Appendix 3) The oral soft tissues were examined and abnormalities noted. The Gingival Inflammation Index [26] was used to assess gingival health, where 6 key teeth (16/55, 21/61, 24/64, 36/75, 41/81, 44/84) were probed with a periodontal probe for presence or absence of bleeding (0 for no bleeding, 1 for presence of bleeding on probing). The teeth were examined after air-drying and a full charting was carried out. The Modified Plaque Index [27] was used to determine oral hygiene status where plaque score (0 for absence, 1 for presence) was recorded on the same 6 teeth. The plaque index was then obtained by adding the number of teeth with

plaque and dividing by the total number of teeth examined. Caries was charted using the WHO criteria. [28]

The FDI Index of Developmental Defects of Enamel (DDE Index) was used for recording enamel defects.[29] Briefly, enamel hypoplasia was recorded either as opacities (diffuse or single), and loss of enamel (either pits, grooves, missing or thinned).

Erosion was charted using the index proposed by Aine et al [22] for classification of tooth erosion caused by GOR. Figure 1 gives examples of teeth showing the different grades of erosion. In this index, grade 0 is recorded where no erosion is present; grade 1 where there is a mild opacity, or etched appearance; grade 2 where the occlusal surface is filled with small holes, the incisal edges are thinned and flattening of cusps is seen on posterior teeth; grade 3 where there is dentin exposure. When a subject had different grades of erosion on different teeth (eg some teeth with grade 1 and some teeth with grade 2) the worst grade of erosion was recorded for that subject.

A saliva sample was obtained by wiping a sterile cotton tipped swab on the patient's tongue for culture of *Streptococcus mutans*. This was then wiped over a culture medium (CRT Bacteria ®) and incubated for 7 days at 37°C. Standard charts for estimation of colony forming units of bacteria were used to read results.

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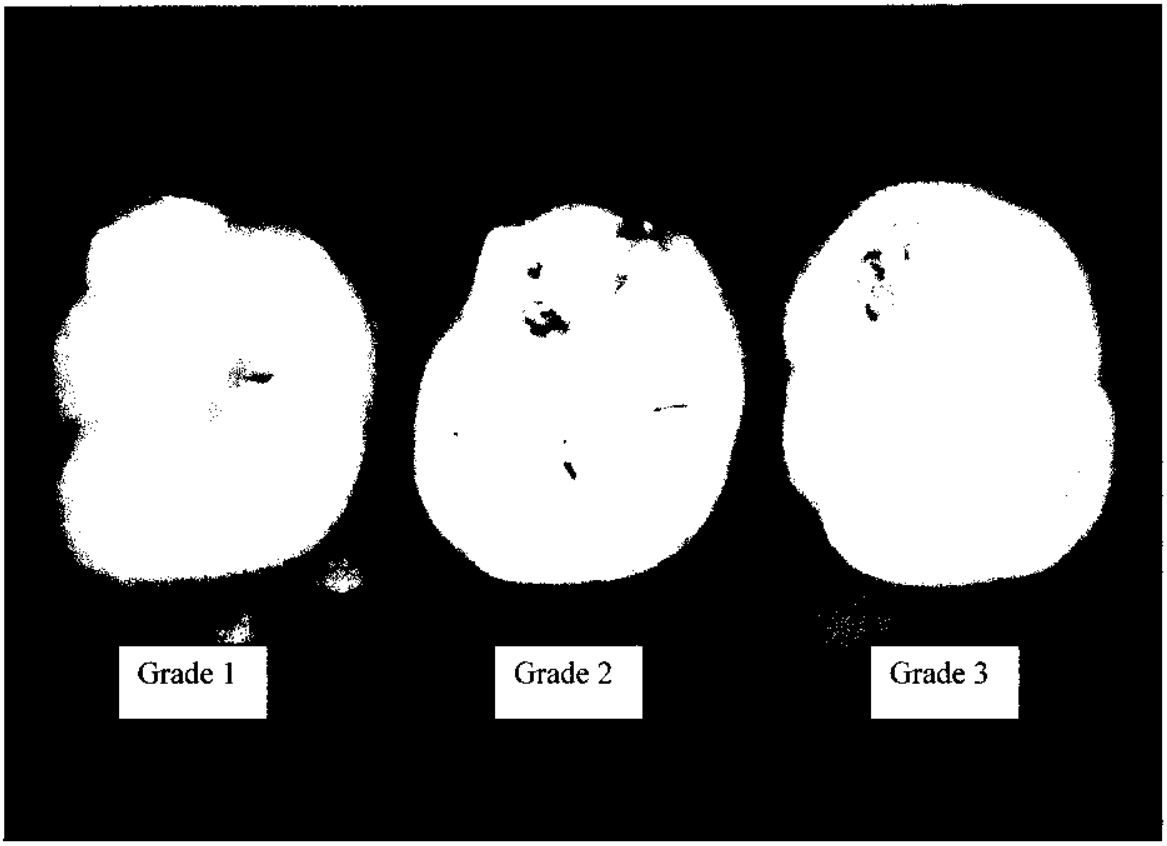

Figure 1: Grades of Erosion

Grade 1: mild opacity, or etched appearance

Grade 2: occlusal surface is filled with small holes, the incisal edges are thinned and flattening of cusps is seen on posterior teeth

Grade 3: dentin exposure.

RESULTS

Demography

Table 1 shows the age and sex distribution of the children examined in the study and control groups. Fifty two study children were examined. In this group there were 31 boys and 21 girls. The mean age was 6.39 years, range (18 months to 15 years 7 months). The control group consisted of 52 children, 27 boys and 25 girls. Age range of controls was 17 months to 15 years 10 months, with mean age 8.56 years. Also shown in Table 1 is the distribution of primary, mixed and permanent teeth. Fifty two percent of children in the study group had a primary dentition compared to 29% of controls; 38% in the study group had a mixed dentition compared to 50% of controls and 5% had a full permanent dentition in the study group compared to 11% of controls.

Oral Hygiene

Plaque Index

Mean plaque index for the GOR subjects was 0.75 (SD = 0.38) and for controls, 0.63 (SD=0.42). As can be seen from Figure 1, the mean plaque index for the control group was lower, but this is not statistically significant. (Fig 2)

Gingival Index

Gingival indices were recorded for both groups, with the GOR group showing a mean gingival index of 0.17 (SD = 0.33) and controls with mean gingival index of 0.13 (SD = 0.26). Although the mean gingival index of the study group was higher than the control, the difference was not significant. ($p>0.05$, Figure 3)

Table 1. Demography of study and control subjects

	Study subjects	**Control subjects**	**Total**
Girls, n (%)	21 (40%)	25 (48%)	46 (44%)
Boys, n (%)	31 (60%)	27 (52%)	58 (56%)
Total	52 (100%)	52	104 (100%)
AGE			
Mean age years (S.D.)	6.65 (3.88)	8.3 (4.12)	7.47 (4)
Girls Mean age years(S.D.) Range years	 5.85 (3.47) 2 to 11.92	 7.26 (3.82) 2.83 to 16.67	 6.55 (3.64) 2 to 16.67
Boys Mean age years (S.D.) Range years	 6.5 (4.1) 1.5 to 15.58	 9.25 (4.2) 1.4 to 15.83	 7.87 (4.15) 1.4 to 15.83
TEETH PRESENT			
No. with primary dentition n (%)	27 (52%)	15 (28.9%)	42 (41%)
No. with mixed dentition n (%)	20 (38%)	26 (50%)	46 (44%)
No. with permanent dentition n (%)	5 (10%)	11 (21.1%)	16 (15%)
Total	52	52	104 (100%)

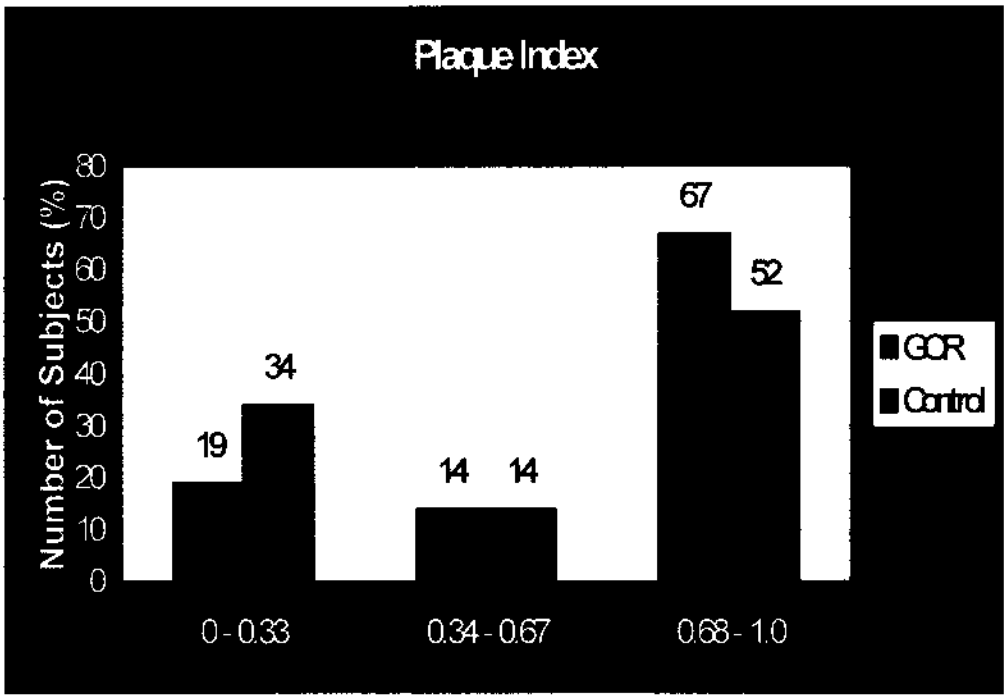

Figure 1: Plaque Index in GOR and Control Subjects

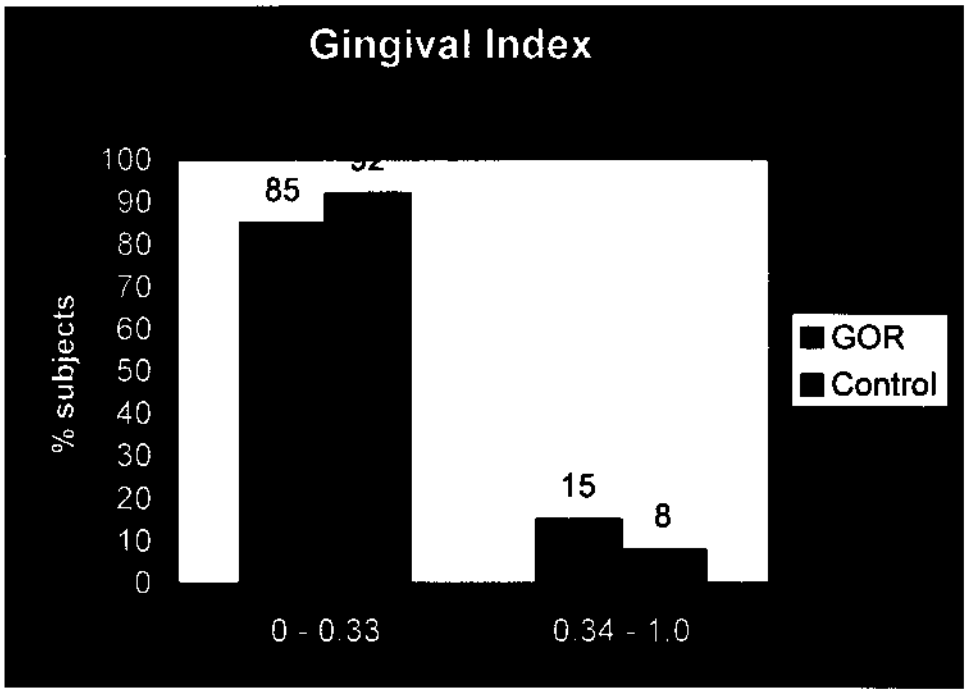

Figure 2: Gingival Index in GOR subjects compared to Controls.

Prevalence of Caries

Subject Prevalence of Caries

There was no difference in subject prevalence of caries experience, with 23 (44%) study subjects and 20 (38%) controls having caries experience. ($p = 0.42$) (Table 2). Also the number of caries free subjects was 29 (56%) in the GOR group and 32 (62%) in the controls ($p = 0.55$)

No significant difference was found in mean subject dmft/DMFT scores, with GOR subjects having a mean of 2.02 and controls having a mean of 1.46 ($p = 0.35$).

Tooth Prevalence of Caries

By contrast, when the total number of decayed, missing or filled teeth (dmft/DMFT) was examined, study subjects had 105 (9.7%) teeth affected compared to 72 (6.2%) in the controls. This difference in subject prevalence dmft/DMFT was statistically significant. ($p = 0.001$)

A significant difference was found between GOR and control subjects when individual numbers of teeth dmf/DMF scores were considered in all three groups (decayed, missing and filled teeth). Of the total number of teeth with dmft/DMFT score, GOR subjects had 52 (50%) decayed teeth compared to 24 (33%) in the control group ($p = 0.01$). GOR subjects had 8 (7%) teeth missing due to caries compared to 1 (1%) ($p = 0.05$) in the controls. Filled teeth numbered 45 (43%) in the GOR group while controls had 49 (66%) ($p = 0.002$). Hence GOR subjects had significantly more decayed and missing teeth due to caries and less filled teeth than controls.

Table 2. Prevalence of caries in GOR subjects compared to control subjects.

	GOR N = 52	**Control** N = 52	**Total** N = 104	**p value**
Subject Prevalence				
Number subjects with caries experience	23 (44%)	20 (38%)	43 (41%)	NS
Number subjects caries free	29 (56%)	32 (62%)	61(59%)	NS
Total number of subjects	52 (100%)	52 (100%)	104 (100%)	
Mean dmft/DMFT (SD)	2.02 (3.4)	1.46 (2.9)	1.74 (3.2)	NS
Tooth Prevalence				
Decayed (d/D) Number of teeth	**52 (50%)** n=105	**24 (33%)** n = 74	**76 (42%)**	**p = 0.01** $x^2 = 7.23$ df = 1
Missing (m/M) Number of teeth	**8 (7%)** n=105	**1 (1%)** n = 74	**9 (5%)**	**p = 0.05** $x^2 = 3.57$ df = 1
Filled (f/F) Number of teeth	**45 (43%)** n=105	**49(66%)** n = 74	**84 (47%)**	**p = 0.002** $x^2 = 9.5$ df = 1
Total Number of teeth (dmf/DMF)	105 (9.7%) N = 1082	74 (6.2%) N = 1193	179 (8%) N = 2275	p = 0.001 $x^2 = 9.7$ df = 1

Erosion Prevalence

Subject Prevalence of Erosion

Table 3 shows the prevalence of erosion in the two groups. Forty six percent of study children exhibit erosion compared to 40% of controls ($p = 0.5$). Although subjects in both groups have more teeth with erosion in the primary dentition than the permanent dentition, differences are not significant between the two groups ($p>0.05$). There is no significant difference in the subject prevalence of erosion in the primary dentition between the two groups, with 45% of GOR subjects having erosion in their primary teeth compared to 46% in the control group ($p>0.05$).

Likewise in the permanent dentition, there is no significant difference in the subject prevalence of erosion with 16% of subjects showing erosion in the GOR group compared to 8% in the control group ($p >0.05$).

Teeth Prevalence of Erosion

When the overall number of teeth affected by erosion between the two groups is considered, there are significantly more teeth in the study group showing erosion than in controls (Table 3). In the study group, 155 teeth were affected by erosion compared to 125 in the control group ($p = 0.005$).

In the primary dentition, there was no significant difference between the number of teeth affected between study and control groups ($p = 0.68$). On the other hand, a significant difference was seen in the permanent dentition, with 14 (4%) teeth affected in the study group compared to 5 (0.8%) teeth in controls ($p < 0.001$).

Table 3: Prevalence of erosion in GOR subjects compared to Controls

	GOR	Control	Total	p value
Subject Prevalence				
Number of subjects with erosion	24 (46%) (N= 52)	21 (40%) (N = 52)	45 (43%) (N = 104)	NS
Number of subjects with erosion in primary teeth	21 (45%) n = 47	19 (46%) n = 41	40 (45%) n = 88	NS
Number of subjects with erosion in permanent teeth	4 (16%) n = 25	2 (8%) n = 24	6 (12%) n = 49	NS
Tooth Prevalence				
Number of primary teeth with erosion	141 (20%) n = 713	120 (20%) n = 580	261 (20%) n = 1293	NS
Number of permanent teeth with erosion	14 (4%) n= 369	5 (0.8%) n = 617	19 (2%) n = 986	p = 0.0009 x^2 = 10.9 df = 1
Total number of teeth with erosion	155 (14%) N = 1082	125 (10%) N = 1197	280 N = 2279	p = 0.005 x^2 = 7.8 df = 1

Severity of Erosion

Number of Subjects with erosion in each grade of severity

Table 4 shows there were a similar number of subjects having erosion in each grade of severity in both GOR and controls ($p > 0.05$). Although there were more subjects with erosion in the severe category (grade 3) in the GOR group than controls, (46% compared to 24%) this was not significant ($p>0.05$).

Severity of Erosion by Number of Teeth

There were a greater number of teeth with erosion in the milder categories (grades1 and 2) in the control group than the study group. In the GOR group, 18 teeth (12%) show erosion grade 1, compared to 25 teeth (20%) in the controls ($p = 0.05$). Erosion grade 2 is also less common in the study group (70 teeth or 45%) than controls (89 teeth or71%) ($p < 0.001$).

However there was a greater number of teeth with grade 3 erosion in the study group, 67 (43%), compared to 11(9%) in the controls ($p < 0.001$).

Table 4: Severity of erosion in GOR subjects compared to control subjects

	Study (N = 24)	**Control (N = 21)**	**Total**	**p value**
Subjects				
Grade 1	2 (8%)	4 (19%)	6 (13.5%)	NS
Grade 2	11 (46%)	12 (57%)	23 (51.5%)	NS
Grade3	11 (46%)	5 (24%)	16 (35%)	NS
Total	24 (46%)	21 (40%)	45 (43%)	NS
Teeth				
Grade 1	18 (12%) n = 155	25 (20%) n = 125	43 (15%) n = 280	p=0.05 $x^2 = 3.74$ df = 1
Grade 2	70 (45%) n = 155	89 (71%) n = 125	159 (57%) n = 280	p<0.001 $x^2 = 19$ df = 1
Grade3	67 (43%) n = 155	11 (9%) n = 125	78 (28%) n = 280	p<0.001 $x^2 = 41$ df = 1
Total	155 (14%) N = 1082	125 (10%) N = 1193	280 (12%) N = 2275	p=0.005 $x^2 = 7.8$ df = 1

Prevalence of *Streptococcus mutans* and Erosion

Twenty two subjects (42%) in the study group had *S. mutans* counts of > 10^5 compared to 13 (25%) in the control group. This difference is not statistically significant but is approaching significance (p = 0.06) (Table 5).

In the GOR group, erosion was found more commonly in subjects with > 10^6 CFU/ml than in subjects with < 10^6 CFU/ml (23% vs 19%). A greater proportion of subjects with no erosion showed lower *S. mutans* counts (35% vs 23%). However, these were not statistically significant (p >0.05).

In the control group, *S. mutans* was less likely to be isolated. When *S.mutans* was found in > 10^6 CFU/ml, more cases were seen with no erosion (21% vs 4%). This may indicate that *S. mutans* is not significantly associated with erosion development. In fact, in the group with lower *S. mutans* counts, erosion was seen almost equally as no erosion (36% vs 38%).

This is reinforced when erosion prevalence and *S. mutans* counts are compared for all subjects. (Table 5). No statistically significant difference is seen (p = 0.6).

Association of *S. mutans* with caries.

When the number of subjects in both groups with *S. mutans* counts of greater than 10^6 were compared with presence of caries, it was found that there was a positive correlation between the isolation of *S. mutans* and caries experience (p = 0.05). (Table 6) In the group with caries, *S. mutans* counts of greater than 10^6 CFU/ml saliva was found in 54% of subjects, compared to 46% of subjects who were caries free. Conversely, 65% of subjects who were caries free had < 10^6 CFU/ml saliva compared to 35% of subjects with caries experience.

Table 5: Presence of *Streptococcus mutans* and erosion in GOR subjects compared to controls

	GOR N = 52				Control N = 52				Total N = 104	
S.mutans CFU/ml	Erosion	No Erosion	Total	p value	Erosion	No Erosion	Total	p	Total	p value
$> 10^6$ *	12 (23%)	10 (19%)	22 (42%)	0.3	2 (4%)	11 (21%)	13 (25%)	0.03	35(33 %)	0.06
$< 10^6$ ¶	12 (23%)	18 (35%)	30 (58%)		19 (36%)	20 (38%)	39 (75%)		69 (67%)	
Total	24 (46%)	28 (54%)	52 (100%)		21 (40%)	31 (60%)	52 (100%)		104 (100%)	

* $>10^6$ CFU/ml saliva = greater than 1,000,000 colony forming units per ml of saliva.

¶$<10^6$ CFU/ml saliva = less than 1,000,000 colony forming units per ml of saliva.

No association between number of subjects with erosion in both groups and *S. mutans* counts of greater than 10^6 CFU/ml ($p > 0.05$).

Table 6: Association of *S. mutans* with caries in all subjects

No. of subjects	> 10^6 CFU/ml*	< 10^6 CFU/ml¶	Total	p value
Caries	19 (54%)	24 (35%)	43(42%)	p = 0.05
Caries Free	16 (46%)	45 (65%)	61 (58%)	df = 3.64
Total	35 (33%)	69 (66%)	104 (100%)	

* >10^6 CFU/ml saliva = greater than 1,000,000 colony forming units per ml of saliva.

¶ <10^6 CFU/ml saliva = less than 1,000,000 colony forming units per ml of saliva.

Prevalence of Developmental Defects of Enamel

Enamel hypoplasia was found in 22 of the study subjects (42%) compared to 8 (15%) of the controls ($p = 0.002$). (Table 7). When total numbers of teeth affected are considered, study subjects had 124 (11.5%) teeth with enamel hypoplasia compared to 49 (4%) of controls ($p= 0.004$). When the GOR group and controls were compared, there was a greater amount of enamel defects in the GOR group in both primary and permanent dentition compared to controls (Table 7). In the primary dentition, 12% of teeth were affected in GOR group compared to 4% primary teeth in controls ($p < 0.0001$). Likewise in the permanent dentition, 10% of permanent teeth were affected in the GOR group compared to 4% in the control group. ($p = 0.0003$).

Table 8 shows the distribution of enamel defects by tooth type. It can be seen that primary incisors are the most frequently affected followed by primary second molars in the GOR group.

Dietary Sugar and Acid Exposure

As can be seen in Table 9, when the three day diet sheets were analyzed, no difference was found in the mean number of sugar exposures (either sugar with meals or in between meals) per day between the groups (3.4 in each group).

Similarly, no difference was found in the mean number of acid exposures (0.7 study, 0.8 control).

Table 7: Prevalence of Developmental Defects of Enamel:

Enamel Hypoplasia	**GOR**	**Control**	**Total**	**x^2**	**df**	**pvalue**
Number of subjects affected n (%)	22 (42%) n = 52	8 (15%) n = 52	30 n = 104	9.2	1	0.002
Primary teeth affected n (%)	87 (12%) n = 713	24 (4%) n = 590	28.05 n = 1303	28	1	< 0.0001
Permanent teeth affected n (%)	37 (10%) n = 369	25 (4%) n = 603	62 n = 972	13.2 5	1	0.0003
Total number of teeth affected N (%)	124 (11.5%) N = 1082	49 (4%) N = 1193	173 N = 2275	49.2 5	1	<0.0001

Table 8: Distribution of Hypoplasia by Tooth Type

Teeth Group	GOR (N = 1082)	Control (N = 1193)	Total (N = 2275)
Primary Incisors	30 (2.7%)	8 (0.7%)	38 (1.7%)
Primary Canines	16 (1.5%)	4 (0.3%)	20 (0.9%)
Primary First Molars	16 (1.5%)	4 (0.3%)	20 (0.9%)
Primary Second Molars	25 (2.3%)	8 (0.7%)	33 (1.4%)
Permanent Incisors	22 (2.0%)	16 (1.3%)	38 (1.7%)
Permanent First Molars	15 (1.4%)	9 (0.8%)	24 (1%)
TOTAL	124 (11.4%)	49 (4.1%)	173 (7.6%)

Table 9: Comparison of number of sugar and acid exposures per day

	GOR N = 36	Control N = 39	Total N = 74	p value
Mean sugar / day	3.4	3.4	3.4	NS
Mean acid / day	0.7	0.8	0.75	NS

DISCUSSION

There is little reported in the literature on the oral health of children with GOR compared to controls. [30] Of the reports that have been published, most deal only with the prevalence of dental erosion in these subjects. This study examines a group of children with GOR compared with a group of healthy siblings. Examination included assessment of oral hygiene, prevalence of erosion, caries, developmental defects of enamel, and frequency of isolation of *S. mutans.* The results of this study show that children with GOR have more dental problems than control siblings. Greater numbers of carious teeth, poorer oral hygiene, more erosion and more than twice the number of teeth with enamel hypoplasia were found in subjects with GOR than the control group.

When the prevalence of dental caries in both groups was examined, although there was no difference in subject prevalence of caries, those subjects who did have caries had greater numbers of affected teeth. Numbers of teeth decayed and missing due to caries were significantly greater in the GOR group, while the number of filled teeth was lower, indicating that there is more untreated caries in this group. This could be due to several reasons. Firstly, parents of children with medical problems may place less importance on dental health, resulting in less attention to oral hygiene as well as not seeking dental treatment for their child. Secondly, difficulties with feeding and failure to thrive could result in more cariogenic diets given to the children in attempts to coerce them to eat.

There are few studies into salivary levels of *S. mutans* in conditions associated with intrinsic acid sources. Jarvinen [24] found no particular differences in salivary microbial counts in patients with gastrointestinal disturbances including GOR. Increased occurrence of mutans streptococci has been found in subjects with bulimic eating disorders. [31] It could be supposed that the resulting environment in the mouth is similar in

bulimics and subjects with GOR. [32] We hypothesize that the acidic environment in the mouth associated with GOR could favour colonization with aciduric bacteria. If subjects with GOR have higher infection rates of mutans streptococci, then it would be likely that caries rates would also be increased. In the present study *S.mutans* was isolated slightly more frequently in the GOR group (42%) compared to controls (25%), which is approaching significance ($p = 0.06$). This could contribute to the higher teeth prevalence of caries in study subjects.

There is very little in the literature on the prevalence of caries in children with GOR. O'Sullivan, [23] examined 53 children aged 2 to 16 years (mean age 4.9) attending two Children's Hospitals in the UK for moderate to severe GOR, finding almost all children were caries free. In contrast, in the present study, only a little over half of all children were caries free. These differences could be due to the younger age group in the UK study.

A few studies have looked at the association of GOR and erosion in children. Aine et al,[22] examined a group of children attending a university hospital pediatric clinic for GOR. Erosive lesions were found in 87% of these subjects. In the study by O'Sullivan [23] erosion was reported in 17% of children with GOR. Only one child was found to have erosion involving dentine, and no child showed erosion in the permanent dentition.

In the present study, greater numbers of teeth with mild erosion (grade 1 and 2) were seen in the control group whereas more teeth with severe erosion (grade 3) were seen in the GOR group. Despite the fact that the number of teeth with mild erosion was greater in the control group, the GOR group had a significantly higher total number of teeth with erosion of any grade.

Erosion is common in both groups with overall prevalence in the study group of 46% and 40 % in the controls ($p > 0.05$). No significant difference was seen in the numbers of subjects with erosion severity of any type. However, when teeth prevalence of erosion was considered, the GOR group had a greater total number of teeth affected. In this study, both groups show equal amounts of erosion in the primary dentition. Primary teeth have thinner enamel which is more prone to acid erosion[33]. Also, the GOR group had greater numbers of permanent teeth affected by erosion than controls.

The present results differ from previous reports. The study by O'Sullivan et al,[23] did not find any erosion in the permanent teeth. Also, only one patient in the O'Sullivan group showed erosion into dentin. In the present study, grade 3 erosion (erosion into dentin) was found in 21% of subjects in the GOR group. One reason for these differences could be that the group examined in the O'Sullivan study was younger (mean age 4.9 years). It has been shown previously that dental erosion occurs only after gastric acid has acted on the teeth several times a week for at least 1-2 years, so that erosion lesions become clinically evident only after a relatively long period of acid exposure. [13]

The erosion seen in the present study is considerably less than the Aine et al[22] study, where erosion was seen in 15 of 17 subjects (87%). There could be a number of reasons the erosion prevalence is much higher than in the present study. Their sample size is comparatively small and it should be noted that two patients who were included in their study had dental erosion which in fact led to the diagnosis of GOR. In addition, there may be other modifying factors not measured in the study which may be involved in whether or not erosion is manifested. It is not known to what extent GOR causes a decrease of oral pH, that is whether or not the gastric contents reach the mouth. Ambulatory pH monitoring in GOR measures the reflux into the distal esophagus. Gudmundsson et al, [34]

found no changes in oral pH during repeated episodes of acid reflux, suggesting that other factors are involved.

The relationship of erosion and *S. mutans* counts has been examined in a previous study. In this study, subjects in both groups with erosion did not have significantly higher S. mutans counts. This is in contrast to the findings of O'Sullivan et al [35] who found a correlation between SM counts and erosion ($p = 0.05$), where children with erosion were compared to children without erosion who were either caries free or caries active. Erosion occurs as a result of acid in the oral environment, either intrinsic or extrinsic. Because the O'Sullivan study group was selected on erosion presence, the acidic oral environment necessary to cause the erosion is hypothesized to favor increased counts of *S.* mutans, as was confirmed by O'Sullivan et al.[35] However in the present study, high *S.mutans* levels were seen in patients both with and without erosion.

In addition, a considerable number of subjects also showed erosion in the presence of low *S. mutans* levels. Although it is hypothesized that *S. mutans* can survive the acidic environment found in reflux, gastric acid pH may be below 3 and at pH lower than 4.2, *S. mutans* cease to metabolize.[36] This could explain the low *S.mutans* levels in patients with severe GOR and erosion.

These findings further suggest that other mechanisms may be involved in the manifestation of erosion. In addition to the question of whether reflux causes simultaneous drops in oral pH, salivary factors such as flow rate and buffering capacity may also play a role . O'Sullivan et al [35] in a study examining risk factors for dental erosion found that low unstimulated salivary flow increased risk of erosion 18 times and low buffering capacity increased erosion risk 14 times. Similar risk factors were found by Jarvinen et al,[21] who reported that an unstimulated salivary flow of less than 0.1ml

/min was associated with a 5 times increased risk of erosion. Gudmundsson et al,[34] observed that significantly more patients with erosion had low buffer capacity compared to controls.

This study also examined the amount of enamel hypoplasia as this may influence the frequency that other oral diseases are seen, such as caries and erosion. Enamel hypoplasia was seen in the study group (42%) more frequently than the controls (15%) ($p = 0.002$). Enamel hypoplasia may be the result of altered gastroenterological function in GOR – eg the altered absorption or increased infection risk which can lead to altered enamel formation. [37] Furthermore, the higher prevalence of enamel hypoplasia may be an aetiological factor in the development of the higher caries rate, [38, 39] and may also contribute to the higher prevalence of erosion.

CONCLUSIONS AND RECOMMENDATIONS

This study shows that children with GOR are at greater risk and have more dental problems than healthy siblings. This includes more untreated caries, more severe erosion and more enamel hypoplasia. Greater emphasis needs to be placed on improving the oral health of these children and to highlight the need for better preventive programs to decrease their risk.

Furthermore, more research needs to be done to assess the extent to which oral pH is changed during reflux episodes and to investigate modifying factors in the erosion process, especially the role of salivary flow rate and buffering capacity. In addition, further investigation of the prevention of erosion is necessary. Investigating preventive

strategies such as the effect of fluoride on erosion progression, the enhancement of salivary defense mechanisms and the role of dietary neutralization of acids would be beneficial in finding ways to prevent erosion in subjects at risk of erosion.

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LIST OF TABLES AND FIGURES

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RESEARCH ROAD
Tel: (07) 3365 4582
Fax: (07) 3365 4455
Email: l.martin@.research.uq.edu.au

APPENDIX 1 ETHICS APPROVAL

THE UNIVERSITY OF QUEENSLAND
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Telephone (07) 3365 3560, 3365 4584
International +61 7 3365 3560, 3365 4584
Facsimile (07) 3365 4455

10/02/99

Dr Vivienne Linnett
Dentistry

Dear Dr Vivienne Linnett

Concerning:-Ethical Clearance for project

Project Title **Oral Health Of Children With Gastroesophageal Reflux**

Project Number **D/17/Dent/98/M**

Your project has been approved by the Dental Sciences Human Ethics Committee, **conditional upon copy of info/consent being given to parents.**

Please note that:-

(I) The Clearance number should be quoted on the protocol coversheet when applying to a granting agency and in any correspondence relating to ethical clearance;

(ii) Clearance will normally be for the duration of the project unless otherwise stated in the institutional clearance:

(ii) Adverse reaction to treatment by subjects, injury or any other incident affecting the welfare and/or health of subjects attributable to the research should be promptly reported to the Head of Department and the Dental Sciences Human Ethics Committee.

(iv) Advisers on 'Integrity in Research'
As part of the University's commitment to the institutional statement, "Code of Conduct for the Ethical Practice of Research" (1990), and the NH&MRC's "Statement on Scientific Practice", designated positions have been appointed as advisers on integrity in research. The Chairperson of each ethics committee acts in an advisory capacity to provide confidential advice on such matters as misconduct in research, the rights and duties of postgraduate supervisors, and procedures for dealing with allegations on research misconduct within the University. The contact number for the Chairperson of each ethics committee can be obtained from the Ethics Officer.

(v) The Committee reserves the right to visit the research site and materials at any time during the project.

(vi) It is the Committees expectation whenever possible, this work should result in publication and the Committee would require details to be submitted for our records.

Staff and students are also encouraged to contact either the Ethics Officer (3365 3924), or Chairperson on other issues concerning the conduct of experimentation/research (e.g. involvement of children, informed consent) prior to commencement of the project and throughout the course of the study.

Yours sincerely

Lynne Martin
Ethics Officer
Office of Research and Post-Graduate Studies

encs.
cc: file

oc Prof W Kim Seow

Dentistry

THE UNIVERSITY OF QUEENSLAND

Institutional Approval Form For Experiments On Humans Including Behavioural Research

Chief Investigator	Dr Vivienne Linnett
Department	Dentistry
Other Investigator(s) /Supervisor	Assoc Prof W Kim Seow
Department2	Dentistry
Project title	Oral Health Of Children With Gastroesophageal Reflux
Project Number	**D/17/Dent/98/M**
Duration	January 1999-June 2000

Granting agency or degree enrolled: Master Dental Science

Comments:

Conditional upon copy of info/consent being given to parents

Name of responsible Ethics Committee:

Dental Sciences Human Ethics Committee

This project complies with the provisions contained in the Council's document 'Statement on Human Experimentation and Supplementary Notes' and complies with the regulations governing experimentation on humans within your institution.

Name of Ethics Committee representative:
Assoc. Prof. Neil Savage
Dept. of Dentistry
Chair, Dental Sciences Human Ethics Committee

Signature N W Savage **Date** 8-02-99

APPENDIX 2

Informed Consent for Study Patients
FOR PARTICIPATION IN A STUDY UNDER
THE UNIVERSITY OF QUEENSLAND SCHOOL OF DENTISTRY AND
THE ROYAL CHILDREN'S HOSPITAL

Project Title: Oral health of children with gastroesophageal reflux.

Investigators:

Dr Vivienne Linnett
MDSc Programme in
Paediatric Dentistry
School of Dentistry
University of Queensland

Dr W. Kim Seow
Associate Professor
School of Dentistry
University of Queensland

Professor Ross Shepherd
Consultant Paediatric
Gastroenterologist
Royal Children's Hospital

You are asked to provide consent for your child to participate in a research study being conducted with the approval of University of Queensland. The following information is provided so you can make an informed consent about your willingness to participate. The purpose of this study is to evaluate the oral features of gastroesophageal reflux. Preliminary studies of other authors have suggested that children with reflux may show more dental problems than unaffected children. Dr. Ross Shepherd, consultant Gastroenterologist at the Royal Children's Hospital has suggested we contact you as your child was scheduled to have an endoscopy procedure.

1. Description of Procedures

The dental examination will be performed at the Dental School. The soft tissues are examined, and any abnormalities noted. The teeth are examined for erosion lesions, dental caries and enamel defects.
Dental photographs may be taken as part of data collection.
No dental xrays will be taken as part of the study.

The levels in the saliva of a bacteria which causes dental caries is assessed. This involves wiping a special plastic strip several times on the tongue.

In addition, a mould (impression) of the upper front teeth will be taken using silicone rubber supported on special trays.

2. Potential Risks and Discomforts

The techniques used for clinical examination are the same as those used for routine dental care. There are no risks involved with the taking of rubber base impressions. Your child will be asleep during the examination.

3. Possible Benefits

The examinations will be rendered free of monetary charge and all findings will be reported to you. Recommendations regarding the need for dental care and referrals to appropriate health care providers will be made when indicated.

4. Alternative Treatment

You understand that this study does not provide for the treatment of any oral conditions which

dentists can be made.

5. Confidentiality
You understand that the information obtained from this study will be kept confidential and that you or your child will not be personally identified in any professional presentations or publications.

6. Withdrawal without prejudice
You can withdraw from this project at any time. Withdrawal will not affect your opportunity to obtain treatment at the School of Dentistry or any other benefits to which you are entitled.

7. Full Committee Review and Executive Clearance
This study has been submitted to the Human Ethics Committee, of the University of Queensland and satisfies the ethical requirements of the National Health and Medical Research Council's guidelines. Whilst you are free to discuss your participation in this study with project staff, Dr. V. Linnett (07 3379 5511) and Dr. K. Seow (07 3365 8061), if you would rather speak to an officer of the University not involved in the study, you may contact the Assistant Ethics Officer (07 3365 4582) or Ethics Officer (07 3365 3924).

Agreement:
Having read these statements, I agree to provide consent for my child in this research project at the University of Queensland School of Dentistry and the Royal Children's Hospital.

_____
Name of Child D.O.B.

_____ _____
Signature of Parent/Guardian Signature of Witness

_____ _____
Signature of Investigator Date

Informed Consent For Control Patients
FOR PARTICIPATION IN A STUDY UNDER
THE UNIVERSITY OF QUEENSLAND SCHOOL OF DENTISTRY AND
THE ROYAL CHILDREN'S HOSPITAL

Project Title: Oral health of children with gastroesophageal reflux.

Investigators:
Dr Vivienne Linnett
MDSc Programme in
Paediatric Dentistry
School of Dentistry
University of Queensland

Dr W. Kim Seow
Associate Professor
School of Dentistry
University of Queensland

Professor Ross Shepherd
Consultant Paediatric
Gastroenterologist
Royal Children's Hospital

You are asked to provide consent for your child to participate in a research study being conducted with the approval of University of Queensland. The following information is provided so you can make an informed consent about your willingness to participate. The purpose of this study is to evaluate the oral features of gastroesophageal reflux. Preliminary studies of other authors have suggested that children with reflux may show more dental problems than unaffected children.
As one of your children is to undergo an endoscopy procedure with Professor Ross Shepherd for investigation of gastroesophageal reflux, we invite the participation of your other child/children as healthy controls in this study.

1. Description of Procedures
For every subject enrolled in the study, a healthy sibling subject matched for sex and closest in age will be randomly recruited from the respective families. Dental examinations of control patients will be done at the University of Queensland School of Dentistry.
The soft tissues are examined, and any abnormalities noted. The teeth are examined for erosion lesions, dental caries and enamel defects.
Dental photographs may be taken as part of data collection.
No dental xrays will be taken as part of the study.

The levels in the saliva of a bacteria which causes dental caries is assessed. This involves wiping a special plastic strip several times on the tongue.

In addition, a mould (impression) of the upper front teeth will be taken using silicone rubber supported on special trays.

2. Potential Risks and Discomforts
The techniques used for clinical examination are the same as those used for routine dental care. There are no risks involved with the taking of rubber base impressions.

3. Possible Benefits
The examinations will be rendered free of monetary charge and all findings will be reported to you. Recommendations regarding the need for dental care and referrals to appropriate health care providers will be made when indicated.

4. Alternative Treatment
You understand that this study does not provide for the treatment of any oral conditions which may be identified during the examination. Treatment needs and referrals to appropriate dentists can be made.

5. Confidentiality
You understand that the information obtained from this study will be kept confidential and that you or your child will not be personally identified in any professional presentations or publications.

6. Withdrawal without prejudice
You can withdraw from this project at any time. Withdrawal will not affect your opportunity to obtain treatment at the School of Dentistry or any other benefits to which you are entitled.

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Agreement:
Having read these statements, I agree to provide consent for my child in this research project at the University of Queensland School of Dentistry and the Royal Children's Hospital.

_____
Name of Child D.O.B.

_____ _____
Signature of Parent/Guardian Signature of Witness

_____ _____
Signature of Investigator Date

APPENDIX 3

Name_____ Sex M / F[90]
DOB_____
Address_____
_____ Postcode_____
Name of Parent / Guardian_____
Phone_____ (H) _____ (W)___
Birth weight_____
Gestational age_____
Height_____
Weight_____

Medical History
Prenatal_____
Perinatal _____
Postnatal_____
Current _____
_____

Medication (Chronic)
Past _____
Current_____

Gastroesophageal reflux
Diagnosed yes/no
Method of diagnosis_____
Aetiology_____
Severity_____
Treatment_____

Dental History_____
_____
_____
Any previous dental diagnosis of erosion_____
Any dental problems previously diagnosed_____
_____
Fluoride
Drops / tablets taken –previous or current_____
Lived in fluoridated area Y / N. How long_____

Diet_____
Breast fed until age_____
Bottle fed until age_____Cow's milk / Soy milk / formula / added sugar
Did child sleep with bottle Y/ N.
Contents of bottle for sleep time_____
Fruit juice use no. of times per day_____
Carbonated drinks no. of times per day_____

APPENDIX 3

Name_____ Sex M / F[90]
DOB_____
Address_____
_____ Postcode _____
Name of Parent / Guardian_____
Phone_____ (H) _____ (W)___
Birth weight_____
Gestational age_____
Height_____
Weight_____

Medical History
Prenatal_____
Perinatal _____
Postnatal_____
Current _____
_____

Medication (Chronic)
Past _____
Current_____

Gastroesophageal reflux
Diagnosed yes/no
Method of diagnosis_____
Aetiology_____
Severity_____
Treatment_____

Dental History_____
_____
_____
Any previous dental diagnosis of erosion_____
Any dental problems previously diagnosed_____
_____
Fluoride
Drops / tablets taken –previous or current_____
Lived in fluoridated area Y / N. How long_____

Diet_____
Breast fed until age_____
Bottle fed until age_____Cow's milk / Soy milk / formula / added sugar
Did child sleep with bottle Y/ N.
Contents of bottle for sleep time_____
Fruit juice use no. of times per day_____
Carbonated drinks no. of times per day_____

APPENDIX 3

Examination

Strep Mutans sample

Soft tissues

Teeth

Plaque 1= plaque present 0= plaque absent

55/16	51/11	64/24
85/46	81/41	74/34

Gingivitis 1= bleeding present 0= bleeding absent

55/16	51/11	64/24
85/46	81/41	74/34

Chart

all restorations present

Caries -decalcified and open lesions including recurrent caries

Enamel Defects/hypoplasia

Score as Opacity (OP) diffuse or single

Loss of Enamel (LE) pits/ grooves/ missing/ thin

Erosion

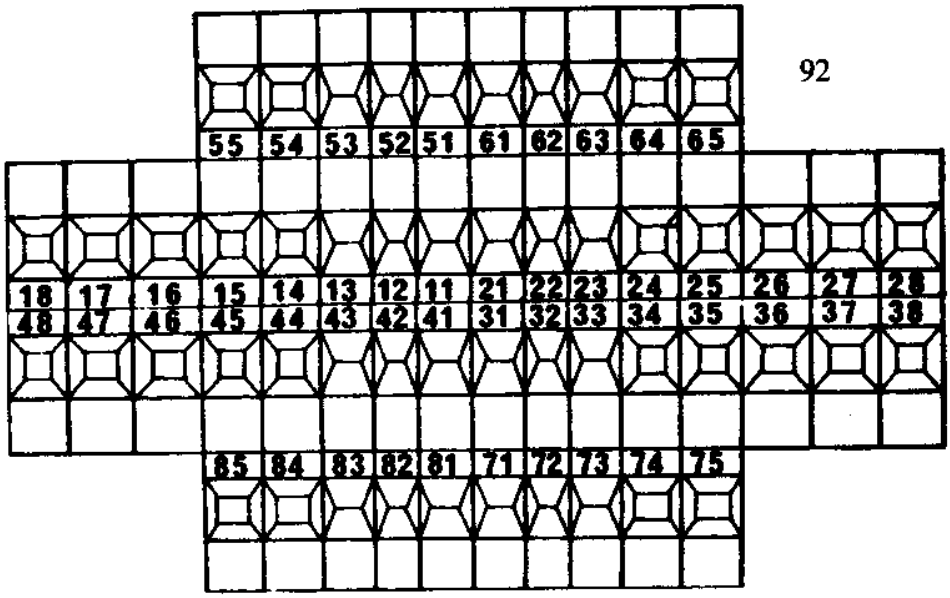
Grade of Erosion	Type of Erosion
Grade 0	No Erosion
Grade 1	Mild opacities or white spots/etched appearance
Grade 2	Occlusal surface filled with small holes(punched out appearance), incisal edges thinned, flattening of cusps.
Grade 3	Dentin exposure at the bottom of the holes on occlusal surfaces or dentin affected on other surfaces

Impressions

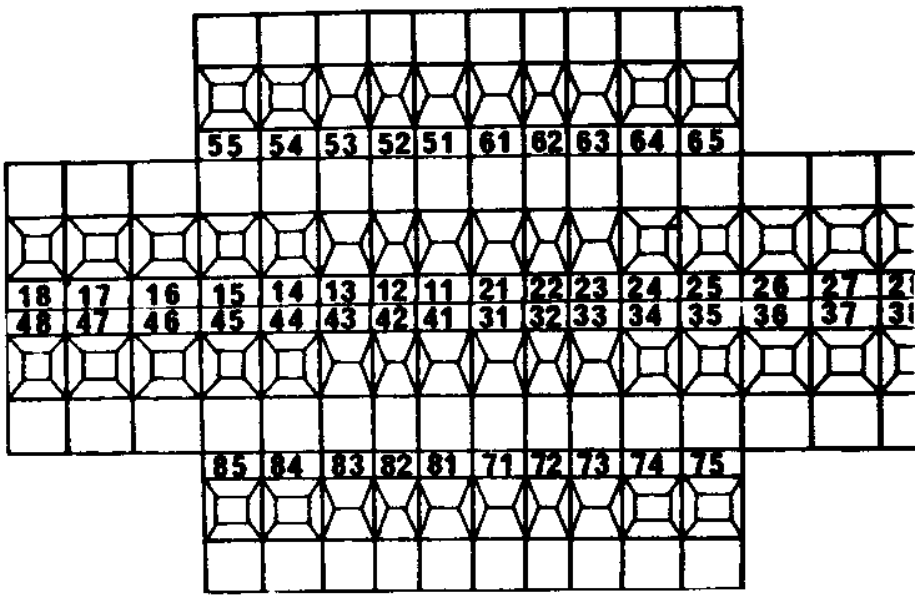
Photographs

APPENDIX 3

Caries
Sound
Decayed
Missing
Filled



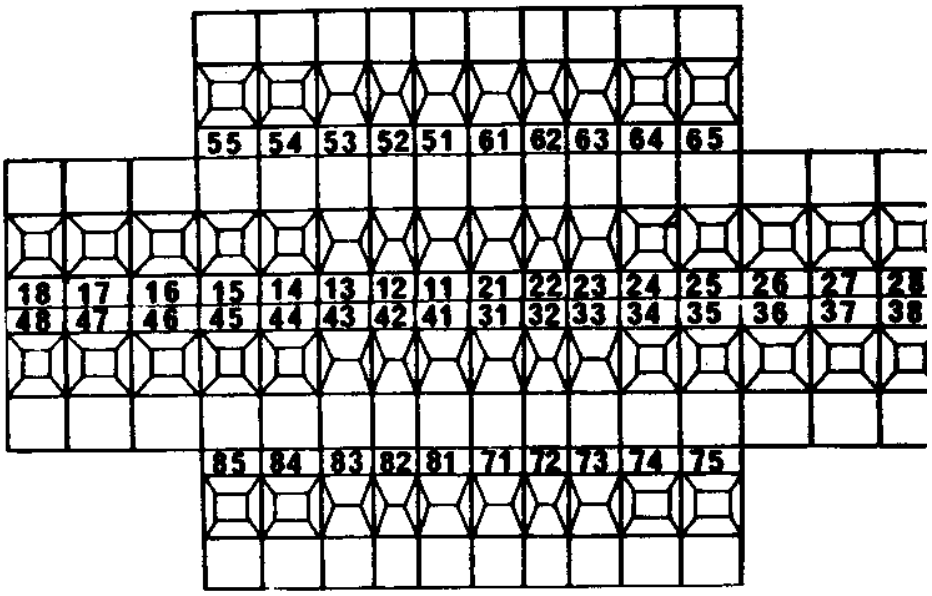
Grade of Erosion	Type of Erosion
Grade 0	No Erosion
Grade 1	Mild opacities or white spots/etched appearance
Grade 2	Occlusal surface filled with small holes(punched out appearance), incisal edges thinned, flattening of cusps.
Grade 3	Dentin exposure at the bottom of the holes on occlusal surfaces or dentin affected on other surfaces



Enamel Hypoplasia

Opacity- diffuse
Single

Loss of Enamel -pits
-grooves
-missing enamel
-thin enamel



ome helpful suggestions in completing this FOOD
)IARY:

. Choose THREE successive days.

'. Make at least ONE of these days a Saturday or a Sunday.

'. Record EVERYTHING that you eat or drink.

'. Provide as much INFORMATION as possible.

'. Describe AMOUNTS in everyday terms such as cup, tablespoon, slice etc.

REMARKS and RECOMMENDATIONS:

		MINIMUM DAILY SERVINGS (MDS)	SERVINGS PER DAY DAY 1	DAY 2	DAY 3	SUMMARY Average	Average minus MDS
MILK and CHEESE	CHILDREN	3					
	TEENS	4					
	ADULT	2					
MEAT and OTHER BODY BUILDERS	EVERYONE	2					
FRUIT and VEGETABLES	EVERYONE	4					
BREAD and CEREALS.	EVERYONE	4					
FAT							
FLUIDS							

SUGAR FREQUENCY		NUMBER PER DAY DAY 1	DAY 2	DAY 3	SUMMARY TOTAL	Average per Day
IN SOLUTION	During meals					
	End of meals					
	Between meals					
IN RETENTIVE FORM	During meals					
	End of meals					
	Between meals					

APPENDIX 4

FOOD DIARY and DIET EVALUATION

PATIENT

DATE

Your Dentist: _____

THE UNIVERSITY OF QUEENSLAND

DENTAL SCHOOL
TURBOT ST.
BRISBANE. Q. 4000

APPENDIX 4

ORE EAKFAST	EAKFAST	TWEEN ALS	NCH	TWEEN ALS	ENING AL	PER

APPENDIX 5

Research Patients Study																	S.Mut			Erosion # teeth									
		m	f	age	d	m	f	dmft	prim	%dmft	D	M	F	DMFT	perm	%DMFT	<106	>106	Plaq	Ging	gr1	gr 2	gr 3	prim	perm	Hypo	Fluoride	sugar	acid
1	Ahquee		Dylon	3.08	1	0	0	1	20	5					0		1		1	0	0					1	0	2.7	0
2	Ahquee		Tiffany	4				0	20	0					0		1		1	0	0					0	0	2.7	0
3	Bartlett		Chris	6	4	0	0	4	18	22.222	0	0	0	0	2	0		1	0.83	0	1	2	1	3		1	0		
4	Blanch		Anthc	2.75				0	20	0					0		1		1	0	0					0	0	4.3	0.7
5	Blanch		Natalie	7				0	12	0	0	0	4	4	10	40	1		0.16	0	0					1	0	4	0.7
6	Brown		Emily	7.67					0	0	2	0	2	4	25	16		1	1	1	1	6		6		1	1	4.7	2.7
7	Buiatta		Anthc	3	0			0	20	0					0		1		0	0	1		20	20		0	0	2.3	0.3
8	Burt		Sami	4	2	0	0	2	16	12.5					0			1	1	0	0					0	0		
9	Burt		Thori	2				0	20	0					0		1		1	0	0					1	1		
10	Byron		Tenille	2				0	20	0					0		1		0.67	0	0					0	0	2	0
11	Byron		Danielle	11					0	0				0	24	0		1	0	0	0					0	0	0.7	0
12	Carlsson		Matth	7.67				0	14	0				0	10	0		1	0	0	0					1	1	3	1.3
13	Chalmers		Scott	5.25		0	0	0	20	0					0		1		0.33	0	1		6	12		1	1		
14	Champion		Patric	13.58				0	6	0					0			1	0	0	0		6			0	0	3.3	0
15	Claxton		Hami	15.83				0	16	0				1	0		1		0.5	0	0					0	0		
16	Clyburn		Lydia	8.25				0	7	0				0	15	0	1		1	0	1		4	4		1	0	3.3	1
17	Coleman		Kimberley	4.25	2	0	0	2	20	10					0		1		1	0	1		8	8		0	0	4.3	0
18	Cornwall		Troy	8	1	1	8	10	15	66.667	0	0	0	0	8	0		1	1	0	1			2		0	1	5.3	1
19	Eccles		Luke	3.75				0	20	0					0		1		1	0	0					1	0		
20	Ennis		Danie	3	1	0	0	1	20	5					0			1	1	0	1			4	4	1	0		
21	Fay		Luke	11	0	0	1	1	3	33.333	0	0	0	0	20	0	1		0.67	0.33	0					0	0	3	1.3
22	Fletcher		Sami	1.42				0	12	0					0		1		1	0	0					1	1	3.7	0
23	Flynn		Hope	2				0	16	0					0		1		0.5	0	0					0	0		
24	Fox		Victoria	11.08	0	0	2	2	4	50				0	20	0		1	1	1	0					1	1	2.7	1
25	Francis		Bryce	3	4	0	0	4	20	20					0			1	1	0	1		5	5		1	0	3.3	1.7
26	Francis		Taryn	3	5	0	0	5	20	25					0			1	1	0	1	4		4		1	0	4	1.3
27	Gill		Carly	11.5	0	0	0	0	5	0				0	19		1		1	0.33	1		2	2		0	1	2.7	0.3
28	Gilliroy		Zach	5				0	20	0					0		1		1	0	0					0	0		
29	Gowlett		Bradl	4.75				0	20	0					0		1		0	0	0					0	0	3.3	0
30	Gowlett		Andre	10.75	0	0	1	1	10	10	0	0	1	1	14	7.1429	1		0.83	0	1	2	7	6	3	0	0	5.7	0
31	Gowlett		Danie	15.58					0		2	0	0	2	28	7.1429	1		1	0.33	1	4	2		6	0	0	5	0
32	Gray		Hayley	3.25	11	0	5	16	20	80					0			1	1	0.16	1		8	8		0	0	4	0.3
33	Greenhalgh		Andre	8.83				0	15	0				0	8	0	1		0.67	0	1	2	8	10		0	1	4.3	0
34	Haase		Kylie	12.5					0					0	28	0	1		0.5	0.5	0					0	0	4.3	0

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35	Kelly	Morg	1		7.67				0	14	0				0	5	0	1		0	0	1		5	7	12		1	1		
36	Lindsay	Katelyn		1	8.58	0	1	5	6	5	120	0	0	1	1	17	5.8824	1		1	1	0						0	0	2.3	0.7
37	MacLeod	Madison		1	3.25	0	0	0	0	20	0				0	0			1	0	0	0						1	0	3.3	3.7
38	Mahony	Josh	1		8.16	3	3	2	8	9	88.889	1		3	4	12	33.333		1	1	0.3	1		1	4	5		0	1	3.7	0.7
39	Martin	Dimit	1		5.5				0	19	0				0	1	0	1		0.83	0	0						0	0	5	0
40	McDougall	Carly		1	5.16				0	16	0				0	8	0		1	0.5	0	0						0	0		
41	Munday	Danie	1		5.5				0	20	0					0		1		0.83	0	1		4		4		1	1	3	2
42	Parmentier	Andr	1		11.33	1			1	1	100	0	0	4	4	23	17.391	1		0	0	1			4		4	0	1	1	1
43	Payne	Hayley		1	2.75				0	17	0					0			1	1	1	1			4	4		0	0		
44	Pool	Alison		1	2.92				0	20	0					0			1	1	0	1		4	10	14		0	1		
45	Reberger	Rayn	1		11.83	3	1	0	4	12	33.333	0	0	0	0	12	0		1	1	0.5	1			4	4		1	0		
46	Tonkin	Ashleigh		1	6.75	1	2	3	6	13	46.154	1	0	1	2	10	20		1	1	0	0						1	1	2.7	0.3
47	Walker	Monique		1	3.75	1			1	20	5				0	0			1	1	0	0						0	1		
48	Uhlmann	Brend	1		6.5	5	0	2	7	16	43.75	0	0	0	0	4	0	1		1	1	1	4			4		1	1	3.3	0
49	Williams	Adria	1		5.5	0	0	0	0	20	0					0		1		1	0.3	0						0	0	4	0.7
50	Williams	Emma-Jar		1	2.25				0	20	0					0		1		1	0	0						1	0	3.7	1
51	Worthingto	Micha	1		12.5				0	2	0	1		0	1	22	4.5455		1	1	0.3	1		1			1	1	0	2.3	1.3
52	York	Jonat	1		12.25				0	0					0	24	0		1	1	1	0						0			

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Research Patients- Control																	S.Mut			Erosion # teeth					En hypo # teeth			
		m	f	Age	d	m	f	dmft	prim	%dmft	D	M	F	DMFT	perm	%DMFT	<106	>106	Plaque	Gingival	gr 1	gr 2	gr 3	prim	perm	0	Fluoride	sugar	acid
1	Addley	Jessica		1	6.17	1	0	2	3	15	20				0	9	0	1		1	0.33	1	4		4		0		2.7	0.3
2	Ahquee	Stephany		1	6				0	18	0				0	2	0	1		1	0	0					0	0	3	0
3	Bartlett	Kassandra		1	4	1	0	0	1	20	5				0	0		1		0.17	0	0					1	0		
4	Blake	Katelyn		1	3.75				0	20	0				0	0		1		0	0	0					0	0		
5	Blanch	Sherryn		1	9				0	4	0			1	1	19	5.2632	1		0	0	0					1	1	2.7	0
6	Bulatti	Briana		1	4				0	20	0				0	0		1		0	0	0					0	0		
7	Byron	Ricky	1		5.17	0			0	12	0				0	12	0	1		0.5	0.16	1	12		12		1	0	2.7	0
8	Cale	Lilly		1	4	0			0	20	0				0	0		1		0	0	0					0		3	0.6
9	Cale	Ned	1		7.92	2	0	2	4	13	30.77				0	10	0	1		1	0	1	6		6		0		3.7	0.3
10	Carlsson	Bianca		1	4.58	2			2	12	16.67				0	10	0	1		0.5	0	0					0	0	2	2.3
11	Casey	Fletcher		1	11.4	0	0	2	2	2	100	0	0	1	1	24	4.1667		1	0	0	0					0	1	3.3	1.7
12	Caslick	Charlotte		1	4				0	20	0				0	0	0	1		1	0.17	1	4			4		1		3.3	1
13	Chalmers	Cherie		1	3.25		0	0	0	20	0				0	0		1		1	0.3	0					0	1		
14	Clyburn	Jake	1		13.6				0	0					0	28	0	1		0.3	0.17	1	3			3	0	1	3.7	1.3
15	Clyburn	Luke	1		15.8				0	0					0	28	0	1		1	0	0					0	1	3	0.7
16	Coleman	Blake	1		8.25		1	1	2	11	18.18				0	10	0	1		1	0	1	1	8	2	11		0	0	4.3	0
17	Coleman	Shaun	1		11.6				0	0					0	24	0	1		1	0	0					0	0	4.7	0
18	Comwell	Nathan	1		3	2	0	0	2	20	10				0	0		1		1	0	1			2	2	0	1	3.7	0.3
19	Comwell	Brendan	1		13	1	0	5	6	12	50	0	0	0	0	12	0	1		1	0	1	1	3	4		0	1	3.7	1
20	Dawson	Rachelle		1	5.17				0	20	0				0	0		1		0	0	0					0	1		
21	Eadie	Louise		1	8.8				0	12	0				0	12	0	1		1	0	1	3	7	10		0			
22	Eadie	stephen	1		11.6				0	8	0				0	16	0	1		0.33	0	1		4	4		1		2.7	2
23	Dowling	Alanah		1	4.58	0	0	5	5	20	25				0	0		1		0	0	0					0	0	15	0.3
24	Elder	Robert	1		13.8				0	0					0	28	0		1	1	0.3	0					0	0	3.3	2
25	Fay	Amy		1	5				0	18	0				0	2	0	1		0.3	0	0					0	0	4.7	1.3
26	Fay	TJ	1		13				0	0					0	28	0		1	1	0.3	0					0	0	4	1.3
27	Flack	Emma		1	13.6	0	0	1	1	2	50	0	0	0	0	24	0		1	1	0.17	1	1		1		0	0	3.3	0.7
28	Fletcher	Rebecca		1	2.83				0	20	0				0	0		1	1	1	0	0					0	0	4	0
29	Fox	Ashleigh		1	8	0	0	3	3	12	25	0	0	0	0	11	0	1		1	1	0					1	1	2.5	0.5

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30	Gill	Matthew	1		5	1	0	0	1	20	5				0	0		1		1	0	1		8		8		0	0	3	1
31	Gill	Royce	1		6				0	14	0				0	8	0	1		1	0	1		9	1	10		0	0	2.7	1
32	Gill	Jeffrey	1		12				0	0					0	28	0	1		1	1	1	2			2		0	1	2.3	0.6
33	Green	Caitlin		1	16.7				0	0	0				0	28	0	1		0	0	1		2			2	0		2.7	2.7
34	Green	Jenna		1	11.8				0	2	0				0	26	0		1	0	0	1		2		2		0			
35	Gowlett	Jeffrey	1		13.8	1	0	0	1	5	20	1	0	4	5	19	26.316	1		0	0	0						0	0	3.3	0
36	Kelly	Taylor		1	6.42				0	20	0				0	0		1		0	0	1		8		8		0	1		
37	MacLeod	Lachlan	1		1.42				0	16	0				0	0		1		0.6	0	0						0	0	3.7	1.3
38	Mahony	Molly		1	6	1	0	3	4	18	22.22				0	5	0	1		0.33	0	1	8			8		0	1	3.3	1.3
39	Mahony	Jacob	1		10.7				0	4	0	0	0	4	4	20	20	1		0.5	0.17	1	2	2		4		0	1	3.7	1
40	Mahony	Luke	1		14				0	0				1	1	28	3.5714	1		0.17	0.17	0						0	1	3.3	1
41	Maloney	Sam	1		9.08				0	12	0				0	12	12	1		0.33	0	1	4	4		8		0	0		
42	Morris	Adam	1		14.3				0	0					0	28	0		1	0.5	0	0						0	1	2.7	0.3
43	Munday	Luke	1		7.5				0	12	0				0	6	0		1	1	0.33	0						0	1	2	2
44	Palbas	Alex	1		3.25	11	0	0	11	20	55				0	0			1	1	0	0						0	0		
45	Parmentier	Danielle		1	13.3				0	0		0	0	1	1	28	3.5714		1	1	0	0						1	1	2.3	0
46	Pool	Cameron	1		4.9	3	0	13	16	20	80				0	0		1		1	0.8	0						0	1		
47	Smith	Damien	1		3				0	20	0				0	0		1		0.6	0	0						0	0	1.3	1
48	Thrum	Michael	1		6.25				0	18	0				0	2	0	1		1	0.17	0						0	1	4	0.3
49	Uhlmann	Kimberley		1	12				0	0					0	27	0		1	1	1	0						0	1	3.7	0
50	Walker	Rebecca		1	7.3				0	14	0				0	6	0		1	1	0	0						1	1		
51	Walker	Scott	1		9.6	1	0	0	1	12	8.333				0	11	0		1	0.8	0	0						0	1		
52	Williams	Richard	1		12.3				0	12	0				0	12	0	1		0.33	0	1		9	3	12		0		2	1